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WASHINGTON, D. C. Revised January 1949

MARKET DISEASES OF FRUITS AND VEGETABLES

POTATOES

Effective November 2, 1953, the responsibility for the work on market diseases of fruits and vegetables was transferred from the former Bureau of Plant Industry, Soils, and Agricultural Engineering to the

AGRICULTURAL MARKETING SERVICE

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UNITED STATES DEPARTMENT OF AGRICULTURE
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MARKET DISEASES OF FRUITS AND VEGETABLES: POTATOES[1]

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[1] An extensive revision and elaboration of the first edition written in 1932 by George K. K. Link, formerly collaborator, Division of Fruit and Vegetable Crops and Diseases, and Glen B. Ramsey. That was the first in the series of Market Diseases of Fruits and Vegetables; this series is designed to aid in the recognition and identification of pathological conditions of economic importance affecting fruits and vegetables in the channels of marketing, to facilitate the market inspection of these food products, and to prevent losses under marketing conditions. The other publications in this series are Miscellaneous Publications No. 121 (tomatoes, peppers, eggplants); No. 168 (apples, pears, quinces); No. 228 (peaches, plums, cherries, and other stone fruits); No. 292 (crucifers and cucurbits); No. 340 (grapes and other small fruits); No. 440 (asparagus, onions, beans, peas, carrots, celery, and related vegetables); No. 498 (citrus and other subtropical fruits); and No. 541 (beets, endive, escarole, globe artichokes, lettuce, rhubarb, spinach, swiss chard, and sweetpotatoes).

The photographs for plate 4, *D* and *F*, were furnished by E. S. Schultz and R. Bonde, plate 9 by R. C. Wright, and plate 20, *D* and *E*, by J. C. Walker, Division of Fruit and Vegetable Crops and Diseases; the photograph for plate 10 by A. H. Eddins, Florida Agricultural Experiment Station; that for plate 11, *A*, by H. O. Werner, Nebraska Agricultural Experiment Station; that for plate 16, *C*, by C. W. Clemmer, Eastern States Farmers' Exchange, Mass.; and that for plate 23, *A*, by D. Folsom, Maine Agricultural Experiment Station.

INTRODUCTION

The potato belongs to the nightshade family (Solanaceae), which includes among many others four species of plants whose vegetative organs or fruits are used as vegetables. The most important of these, the potato (*Solanum tuberosum* L.), is also the most important of all vegetable crops. The others, in order of their importance, are the tomato (*Lycopersicon esculentum* Mill.), the pepper (*Capsicum frutescens* L.), and the eggplant (*S. melongena* L.).

The potato tuber of the market is a storage organ that develops from a swelling at the end of a rhizome, or underground stem. The potato tuber is therefore a fleshy underground stem. The so-called new, or early, potatoes of the market are tubers harvested before they have attained full size and maturity. They have a poorly developed, thin, tender skin, which is easily rubbed off (feathered) during harvesting and handling. Such tubers discolor and wither rapidly at skinned places and are more susceptible to decay than mature tubers. For this reason the early crops are not suitable for long storage.

The late, or main, crop of potatoes, which comes chiefly from the Middle Atlantic and Northern States, is not harvested until the vines are mature or dying and the tubers have a well-developed, firm, tough, corky skin. These tubers are not easily injured; if free of serious diseases and stored under proper conditions (36° to 40° F. and 85 to 90 percent relative humidity), they change very little within 3 to 6 months.

Because many people handle potatoes as if they were dead, it is fortunate that both immature and mature tubers have a remarkable ability to develop protective tissues (to heal) when wounded. Under moderate temperature and humidity conditions the cells about and underneath injuries make chemical and physical changes which render them impervious to water and resistant to infections. The walls of the cells become suberized (corky) and additional layers of new corky cells (wound periderm) are formed under and about the wound. This stops loss of tissue moisture and affords as much protection against decay-producing organisms as did the original skin (periderm).

Under optimum conditions this corky protective tissue develops within 24 to 48 hours after the injury occurs. However, at temperatures of 50° F. and below and at those above 95° suberization (healing) does not take place rapidly enough to offer much protection against wound parasites. For this reason it is advisable whenever possible to hold newly harvested potatoes at moderate temperatures and at as high a humidity as possible for a few days to permit wound suberization before storage at low temperature.

The quality of potatoes depends not only upon the variety and maturity but also upon the manner in which they are harvested and handled immediately after harvest. Tubers that are badly cut and bruised during harvest become discolored, wither, and decay much more rapidly than uninjured tubers. This is particularly true of early potatoes harvested during hot weather. Heat injury (scald) of tubers left exposed in the field before picking up often causes great damage to the early-potato crop. Extreme heat causes death of cells

with subsequent break-down and decay by various organisms; sublethal temperatures predispose the tubers to infection by soft rot bacteria (p. 5).

Sometimes part of the northern late-potato crop is injured by low temperatures before and during harvest. Mild or severe types of freezing injury also predispose tubers to bacterial soft rot when they become warm. Unless tubers injured by freezing are graded out before storage, they may be invaded by any one of several decay-producing organisms. Handling operations that avoid mechanical injuries and extremes of temperature are necessary, therefore, in order to maintain good-quality potatoes and reduce losses.

During transit and storage it is not necessary to refrigerate potatoes at very low temperatures. In fact potatoes are of better cooking quality when they are held at 50° to 60° F. than at 36° to 40° as usually recommended for storage. However, the latter temperature range is advisable for long storage periods in order to hold in check sprouting and development of decay.

(See *3*.) [2]

ALTERNARIA TUBER ROT (EARLY BLIGHT)

(*Alternaria solani* (Ell. and G. Martin) Sor.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Alternaria tuber rot may occur in potatoes from any region because the causal fungus is present to some extent wherever potatoes are grown. However, most of the serious tuber infection occurs in the northern-grown crop when the potatoes are harvested and stored during cool, humid weather. Alternaria tuber rot is a serious storage and market trouble, which often opens the way for secondary infections by species of *Fusarium* and other organisms.

The lesions on the potato tubers are of irregular size and shape, but they seldom cover extensive areas unless several coalesce. Most lesions are superficial and vary from ¼ to 1 inch in diameter. Externally the affected spots are brown to purplish brown and slightly sunken with regular borders (pl. 1, *A*). They appear somewhat similar to those produced by late blight, but internally they are shallower and are sharply set off from the healthy tissues by a layer of cork (pl. 1, *B*). Furthermore, the decay does not feather out irregularly into the healthy tissue as does that of late blight. A yellowish discoloration of the flesh is sometimes produced just beyond the edge of the lesion. Ordinarily no mold or spore production is evident over the lesions on the tubers.

The early blight stage of this disease is characterized by oval to irregular brown to brownish-black spots on the leaves. The young spots, 1/16 to ¼ inch in diameter, are dark brown; the older ones, ⅜ to 1 inch in diameter, are usually lighter brown and marked with light- and dark-brown concentric zones, which produce the so-called target-board effect. Under conditions favorable for the fungus the lower leaves gradually die and dry up until only a few spotted green leaves remain at the top of the plant. In severe cases dark-brown lesions develop on the petioles and upper part of the stems and the olivaceous-black fungus becomes evident on the surface of the diseased areas.

[2] Italic numbers in parentheses refer to Literature Cited, p. 50.

CAUSAL FACTORS

Alternaria solani, the fungus which induces this disease, overwinters in the form of mycelium and spores in plant trash, in the soil, and on potato tubers. The spores produced on the early blight lesions of the new crop are readily disseminated from plant to plant in the field by wind, rain, and insects. The spores are produced most abundantly when rains or dews are frequent and when the temperature is about 78° F. In culture the fungus grows best between 77° and 85°, but it makes no growth below 35° or above 112°.

Most infections on potato tubers occur immediately after harvest. Spores from the blighted vines contaminate the tubers during harvesting and handling operations, and under favorable temperature and moisture conditions they germinate and invade the tubers. Uninjured tubers as well as bruised ones are subject to infection. Wounded tubers are most likely to develop decay under moist conditions at temperatures between 58° and 62° F. Under dry conditions at harvest and during storage, infections are held to a minimum and spread of the disease to unwounded tubers is not likely to be important. Development of decay is very slow at 41° to 45°, most rapid at about 60°, and practically inhibited at 77°.

CONTROL MEASURES

Potato tubers allowed to mature in the ground are less likely to develop alternaria tuber rot than are immature ones. During harvest the vines should be kept away from the tubers insofar as possible, and they should never be piled over tubers or over containers in the field. Excessive wounding of tubers during harvesting and handling should be avoided.

Early blight on potato plants can usually be successfully controlled by thorough and regular spraying with bordeaux mixture. Dithane combined with zinc sulfate has also given good control in some preliminary tests.

(See *21, 61, 77, 97.*)

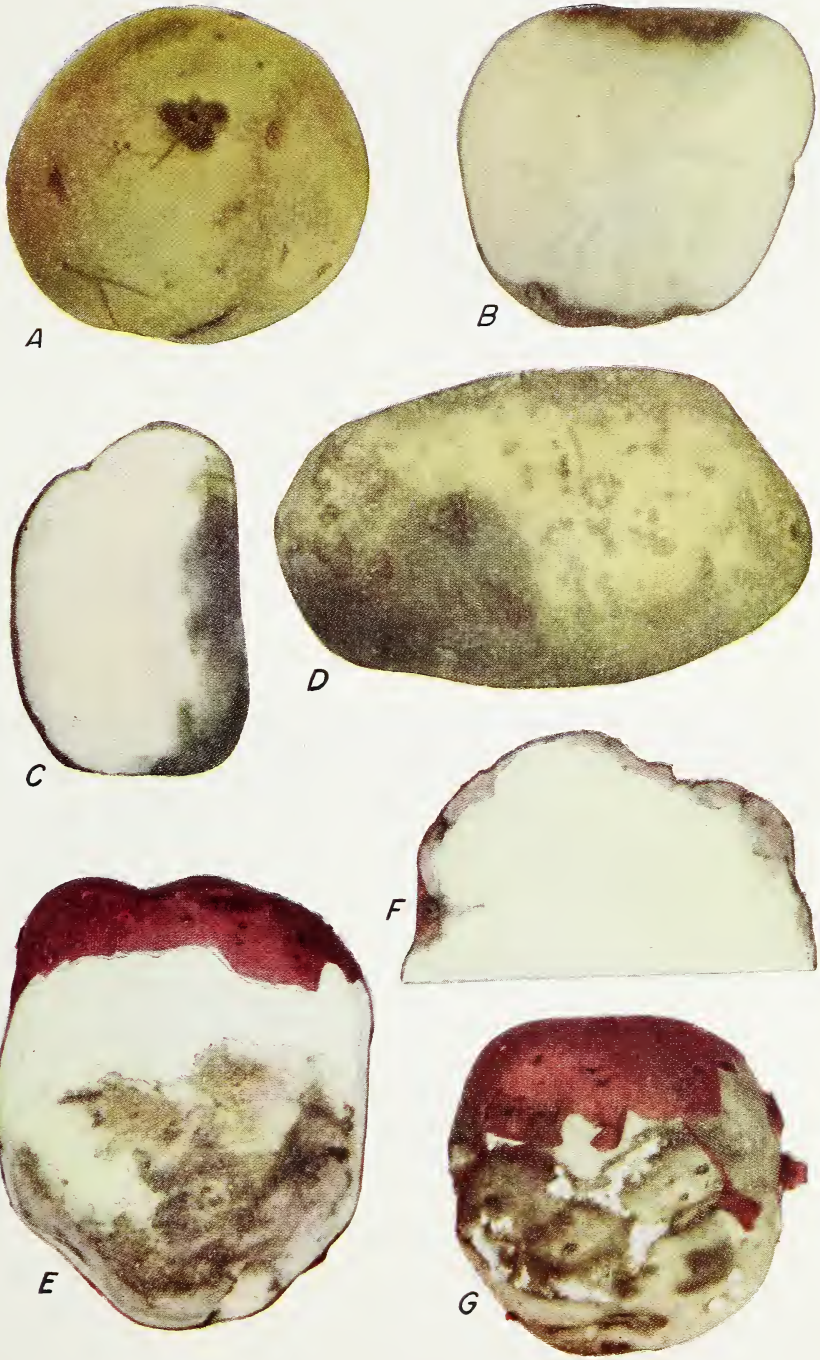
ARMILLARIA ROT

(*Armillaria mellea* Fr.)

Armillaria rot of potatoes has been reported from Washington and British Columbia, where it is a factor only when potatoes are planted on newly cleared land rich in humus. It has also been reported from Australia.

Armillaria mellea, the fungus which induces this rot, furnishes the diagnostic characters of the disease. It forms structures known as rhizomorphs, from which mushroomlike bodies arise. Because of their color these have given rise to the name honey fungus.

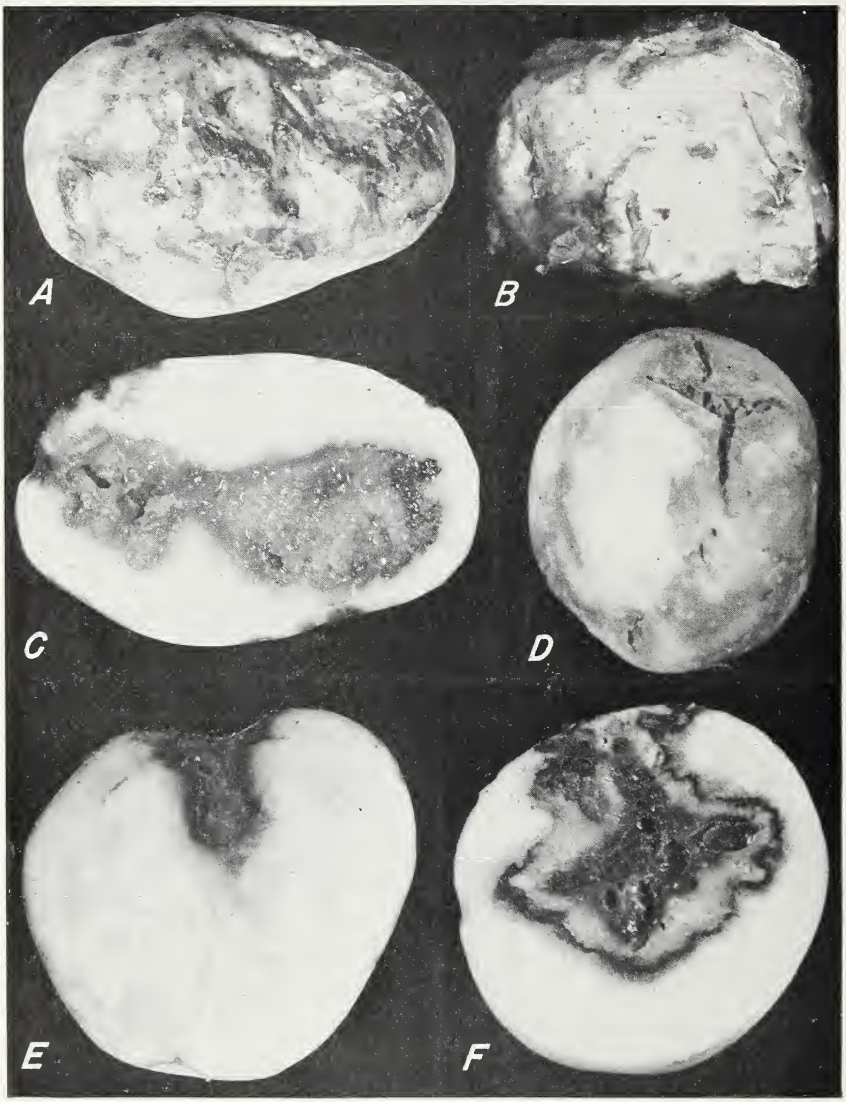
The dark-brown to black rhizomorphs grow about the roots and penetrate the corky layer of the tuber. They are generally attached to the affected areas and may be found adhering to the skin at various points. Affected tubers often show shallow, hard, brown, roughened, shrunken, corky areas of varying extent. The diseased internal tissues are light brown and interspersed with convoluted white, brown-edged mycelial plates.



Potato diseases: *A* and *B*, Alternaria tuber rot; *C* and *D*, pink rot in white variety; *E*–*G*, pink rot in the Red Warba variety.

Wet and moldy sacks from a carload of southern potatoes affected with bacterial soft rot and sclerotium rot.

Culls from a carload of early potatoes affected with bacterial soft rot when unloaded on the Chicago market.



Potato diseases: *A–C*, Bacterial soft rot (advanced stages); *D–F*, blackleg rot.

Armillaria rot can be avoided by planting corn or summer crops instead of potatoes in newly cleared humus soil until the causal fungus is starved out.

(See *5, 17, 95.*)

BACTERIAL SOFT ROT (SLIMY SOFT ROT)

(*Erwinia carotovora* (L. R. Jones) Holland)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Bacterial soft rot, which occurs in potatoes from all regions, probably causes more loss in the early and intermediate crops than do all other potato diseases combined (pls. 2 and 3).

Soft rot may occur as either a primary or a secondary rot. As a primary rot it is found most abundantly in the early crop when the tubers are badly bruised and skinned, when they show heat injury (scald) and enlarged lenticels, and particularly when they are loaded while hot or wet and shipped under insufficient refrigeration (pl. 4, *A–C*, and pl. 5). In late potatoes this decay is most important in tubers showing field freezing. However, late potatoes harvested from subirrigated soil or those harvested and stored under high-temperature conditions may also show wound and lenticel infections similar to those in the early crop (pl. 5, *G*). As a secondary rot soft rot may follow other diseases, especially late blight tuber rot, blackleg, fusarium tuber rot, leak, brown rot, and ring rot. In the field it also often becomes important in wet soils as the cause of seed-piece decay.

The decay of tubers following heat injury and bruises is at first white to cream-colored, but after exposure to air it may become brown to black (pl. 4, *A–C*, and pl. 5, *A*, *C*, and *D*). The affected tissues are generally soft but not watery. The boundary between the soft, disintegrated and the sound, firm tissues is rather sharp. Often the outer parts of the tuber remain intact while the inner ones may be completely destroyed. As the soft tissues dry they become slimy; and finally, when completely dry, they may be reduced to chalky, white masses. Tubers in advanced stages of decay are usually invaded by secondary organisms; as a result of such invasion the decaying tissues become slimy and foul smelling. At this stage the decay is commonly referred to as slimy soft rot (pl. 4, *A* and *B*).

Lenticel infections are characterized in the early stages by water-soaked areas around the lenticels, often with noticeable swelling, up to ¼ inch in diameter. The tissue in these areas is usually firm, and soft rot is rarely observed at this stage (pl. 5, *B*). The depth of the infected areas varies from 1/25 to ¼ inch. If tubers bearing infected lenticels are allowed to remain in a fairly dry atmosphere for several days, the diseased areas lose their turgidity and the centers become slightly sunken (pl. 5, *F* and *G*). As further drying occurs, the tissue beneath the epidermis may become so sunken as to form a pocket, with the epidermis often remaining intact about it. At this stage the epidermis over the lesion is often grayish white with a darker border, and the lenticel in the center still is visible.

If tubers bearing numerous lenticel infections are placed in an environment of high relative humidity and high temperature, soft rot may develop. On the other hand, serious decay is unlikely to occur at the lenticels if the infected tubers are placed in an environ-

ment of low relative humidity and moderate temperature—the conditions often encountered in temporary storage or on the retail market.

It has been commonly observed that potatoes dug from waterlogged soil often have greatly enlarged lenticels (p. 14). Many of these lenticels are invaded by bacteria while the potatoes are still in the soil. During the washing and handling processes, the proliferated tissue may be rubbed off; at such points also bacterial infection may occur (pl. 5, *E*).

CAUSAL FACTORS

Erwinia carotovora attacks a wide variety of truck crops in addition to potato tubers. This bacterium is so abundant in most soils that there probably are no vegetable crops on which it is not present, because of the washing, splashing, and blowing of soil onto them. Furthermore, during harvesting and packing the plant tissues are also contaminated with these bacteria by means of soil, tools, hands, or wash water. Not only are the bacteria present as contaminants in the field, but they cause active rots there and much of the rot that develops in transit and storage has its actual inception in the field.

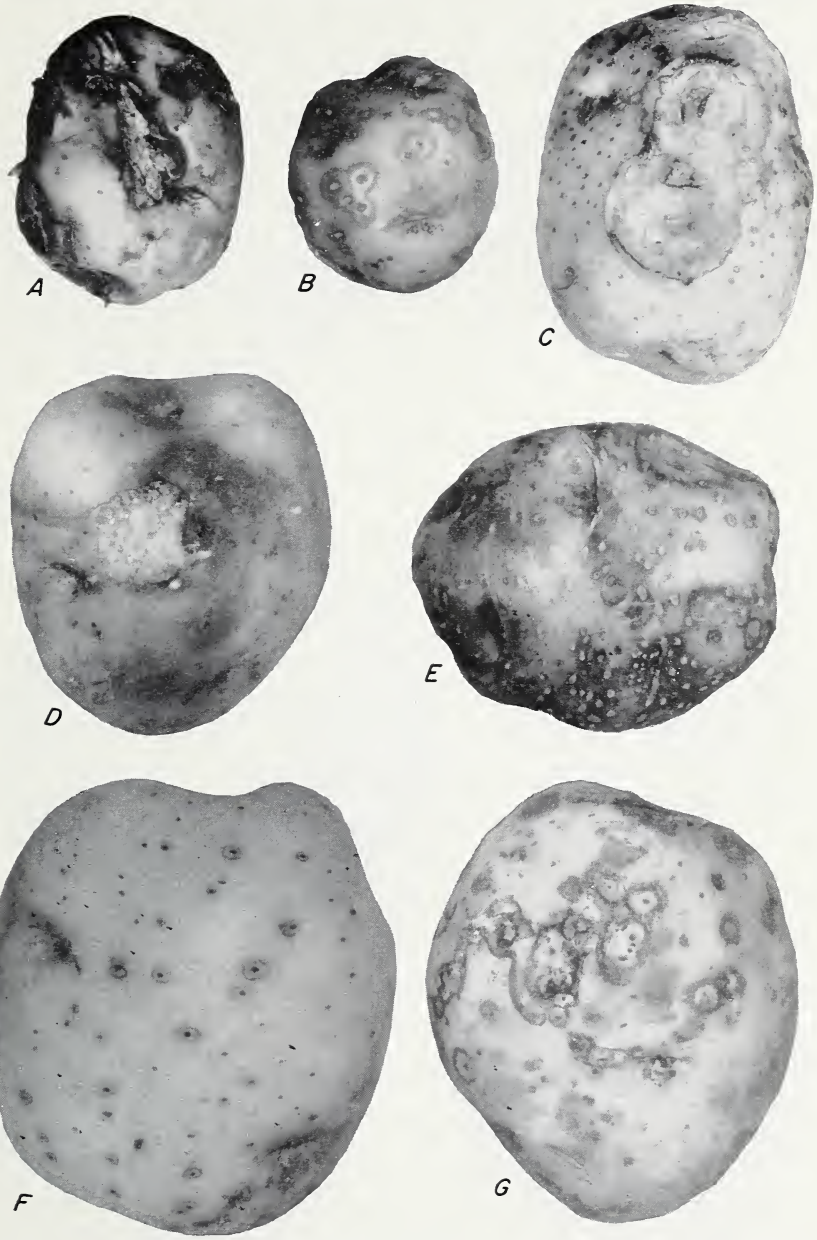
Since the soft rot bacteria invade potato tubers chiefly through injuries, practically all infections occur in tissues weakened or killed by heat injury, freezing, and mechanical injury, in enlarged lenticels, or in lesions caused by other diseases. Soft rot almost invariably appears in potato tubers injured by heat in the field during harvesting and loading (p. 21). Not only killing temperatures (122° F. and above) but also sublethal temperatures (109° to 113°) have been found to predispose potato tubers to soft rot. It should be remembered, however, that such temperatures can be built up in potato flesh when air temperatures are no higher than 90° (p. 21). Both field and laboratory tests have shown that potatoes dug, hauled, graded, and sacked while hot are much more likely to become infected and eventually to decay than similar potatoes handled at moderate temperatures. Even potatoes that have been subjected to only sublethal temperatures have been found to develop less soft rot if they are not handled until after they become cool again.

Erwinia carotovora may cause decay in potatoes at temperatures ranging from 46.5° to 92° F., but ordinarily comparatively little rot occurs below 70°. It has been demonstrated that there is little chance for infection by this organism through either wounds or lenticels in very dry air. Infection is readily obtained, however, at higher relative humidities (94 to 100 percent) and at temperatures of 60° to 85°.

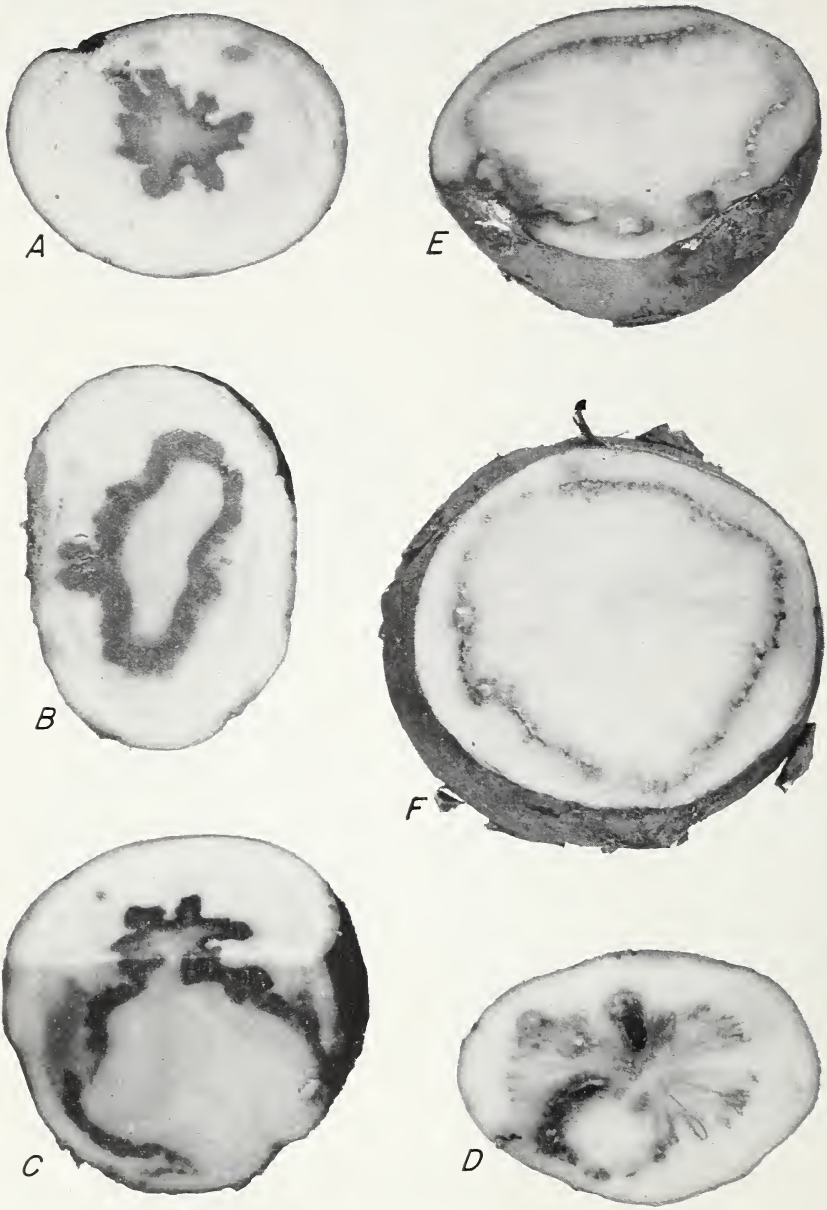
CONTROL MEASURES

As with many other storage diseases, the control of bacterial soft rot begins in the field. The control of field diseases such as late blight, brown rot, ring rot, or blackleg, which produce lesions in tubers through which soft rot infection may occur, is one important step. Furthermore, the protection of tubers during harvest against bruising and exposure to excessive heat or to freezing temperature is of primary importance. Rotting tubers should be sorted out in packing; however, they should not be thrown on manure or compost heaps to be returned eventually to the field. If possible, potatoes that have been washed should be dried before being stored or shipped.

Ventilation and low temperature during transit and storage are important factors, of course, in the control of bacterial soft rot.



Potato tubers affected with bacterial soft rot: *A*, Following mechanical injury; *B*, in lenticels (swollen stage); *C*, following bruise and through lenticels; *D*, soft, watery, gaseous stage following heat injury (scald); *E*, following enlarged lenticels; *F*, in lenticels (flat stage); *G*, in lenticels (northern-grown late potatoes).



Potato diseases: *A–C*, Blackheart; *D–F*, brown rot.

Storage pits, bins, and houses should be so constructed as to provide temperatures between 36° and 40° F. and good ventilation. It has been shown that refrigeration of early potatoes during transit in hot weather is beneficial, since the decay does not make much progress if potatoes are kept at temperatures below 70°.

(See *13, 29, 79, 89, 115, 143, 144, 157, 165, 167, 172, 189.*)

BLACKHEART

Blackheart is an important storage, transit, and market disease of potatoes. It is not known to occur in growing tubers except in flooded soils, where it may occur occasionally in tubers dug during excessively hot weather from wet, waterlogged fields. It has been reported that large potatoes are more susceptible to blackheart than small ones. Blackheart often occurs in transit when the temperature in heated cars is allowed to go over 90° F. It occurs also in storage houses and pits if the piles of tubers are so large that the interior cannot be well aerated; under such conditions it may even occur in refrigerated rooms, especially if the tubers are hot and wet when stored.

External symptoms of blackheart may or may not be evident. In advanced stages of break-down there may be seen on the surface moist areas which are purplish at first but turn brown or black within a short time. The internal symptoms are dark-gray to purplish or inky-black discolorations. Freshly cut tissues may appear normal in color, but after a short exposure to air they turn pink, then gray or purplish, and finally jet black (pl. 6, *A–C*). Usually the affected tissues are sharply differentiated from the healthy ones; they are firm or even leathery if they have dried to any extent. The discoloration is usually restricted to the central part of the tuber, but it may extend to the surface. In advanced stages the affected tissues may dry out and separate, thus forming cavities. When one side of the tuber is exposed to high temperature, as in a heated car or in a sunny field, the discoloration may affect only the exposed side.

Blackheart is caused by a deficiency of oxygen which results in the death of the tissues of the potato tuber. The disease may be brought about at any temperature by an absolute lack of oxygen or as a result of inadequate aeration; or it may develop in a full supply of air if the temperature is so high that the amount of oxygen needed by the tissues is greater than that supplied by the normal air through the outer tuber tissues.

Blackheart has been induced experimentally on tubers held at a temperature of 70° F. in a reduced oxygen supply. No appreciable amount was observed, however, until a temperature of 86° was maintained.

Since tubers given an adequate supply of oxygen will not usually develop blackheart in commercial shipments at temperatures below 90° F., control involves the maintenance of a lower temperature and adequate ventilation. In heated cars or in storage, tubers nearest the source of heat should be protected by tin sheeting. In heated cars the temperature should be kept at about 70°. Tubers should not be left long in hot, light soils after the vines are dead, and they should not be left lying near the surface of the soil or on it after digging during hot weather.

(See *10, 11, 12, 46, 183, 184, 196.*)

BLACKLEG

(*Erwinia phytophthora* (Appel) Bergey et al.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

The disease known as blackleg of potatoes occurs in practically all parts of the United States. It affects all parts of the plant. It is important not only because it kills the growing plant and thus reduces the yield but also because it rots the tubers in the field, in transit, and in storage. Furthermore, blackleg lesions open the way for other bacterial soft rots (p. 5).

The disease derives its name from the jet-black lesions produced at the base of the affected stems. Plants affected with blackleg are characterized by a yellowing of the leaves from the margin inward, an inhibition of branching, and a tendency for lateral stems to stand more erect than those of normal plants. An attacked plant may be dwarfed and die without producing tubers. If infection takes place later, a crop of tubers may be produced but some of them may become infected through the stolon from the mother plant. In wet and heavy soils tubers may be completely destroyed before harvest. It is believed that extensive rotting of tubers may be caused by the entrance of the organism through lenticels in naturally wet or irrigated soils.

The tubers harvested from plants affected with blackleg may or may not show definite symptoms of decay. The first symptoms are usually softening and slight discoloration of the vascular tissues at the stem end of the tuber. The decay is white or grayish at first, but on exposure to the air it eventually becomes dark (pl. 4, *E*). In advanced stages (pl. 4, *D* and *F*) the disease involves the central part of the tuber, converting it into a soft, buttery rot and leaving only a shell. In the long western varieties of potatoes blackleg may appear as a soft, sunken, or shriveled type of stem end rot.

CAUSAL FACTORS

Extensive cultural studies have shown that the blackleg organism, *Erwinia phytophthora*, is probably identical with the soft rot bacterium *E. carotovora*;[3] at least they are very similar.

The blackleg organism overwinters in potatoes in storage and is carried into the field chiefly through the use of infected tubers for planting. The bacteria also are frequently spread from diseased tubers to sound ones by the cutting knife during the preparation of the seed pieces. This occurs more commonly than is suspected, because infected tubers are not necessarily discolored or decayed.

It has been shown that the blackleg organism may overwinter in the soil, and there are indications that irrigation water may carry it from hill to hill. An abundance of moisture and moderately high temperatures are essential for extensive development of the disease in the field.

The seed-corn maggot (*Hylemya cilicrura* (Rond.)) and the seed-potato maggot (*H. trichodactyla* (Rond.)) play an important part in the infection of cut seed pieces both before and after planting. If cut

[3] In the present publication the writers have followed the classification of Bergey and others (*13*), in which *Erwinia carotovora* and *E. phytophthora* are considered to be separate species.

Potato diseases: *A* and *B*, Jelly-end rot; *C* and *D*, browning.

pieces are planted in excessively wet soil, there is little or no development of wound cork. As a result, these seed pieces become infected by various bacteria in the soil. These decaying seed pieces attract the yellowish-white legless maggots if they are not already present, and they burrow over the surface and through the seed pieces. The eggs of these insects are often contaminated with the blackleg organism, which eventually gets into the intestinal tract of the larvae and is thus carried into the tuber tissues by the maggots.

CONTROL MEASURES

The best control of blackleg is obtained by the use of certified seed stock or of seed potatoes from fields known to be free of blackleg. Tubers from infected plants should not be used for seed even though they appear sound. If an infected tuber is accidentally cut during the preparation of seed, the contaminated knife should be disinfected before further use. In order to disinfect the surfaces of tubers that may have become contaminated with the blackleg organism through contact with diseased tubers in handling or in storage, it is often advisable to treat all potatoes with a good disinfecting solution. For control of the tuber rot in transit and storage the discussion of bacterial soft rot (p. 5) should be consulted.

(See *2*, *23*, *24*, *99*, *106*, *107*, *108*, *109*, *110*, *139*, *168*, *181*, *188*.)

BROWNING

"Browning" is the term used to describe the discoloration of skinned areas on immature tubers (pl. 7, *D*). Although some browning occurs eventually on any injured tissue exposed to the air, it is most serious on skinned tubers that are exposed to the drying effect of winds in the field or the packing shed, especially if the weather is very warm. Air movements in loaded cars may also contribute to this type of discoloration.

Slight browning is not objectionable except when large areas are involved and excessive withering results from loss of moisture. Severe browning affects the appearance and therefore the market value of potatoes. If browned potatoes are shipped or stored wet they are predisposed to a moist, sticky, bacterial growth that develops over the surface of the spots and to surface development of molds caused by *Fusarium* (pl. 7, *C*).

Prompt picking up and hauling, protecting tubers from the dry atmosphere in the field by tightly woven bags, covering loaded trucks with tarpaulins, and protecting stacks of potatoes at sheds from sun and wind that cause heat injury and drying have all been found useful in preventing damage to tubers from browning before shipping.

(See *7*, *146*, *157*, *166*.)

BROWN ROT (SOUTHERN BACTERIAL WILT)

(*Bacterium solanacearum* E. F. Sm.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Brown rot is one of the diseases that may attack all varieties of potatoes with varying degrees of severity. In addition it occurs on eggplant, pepper, tomato, tobacco, peanut, and many other plants. It is primarily a disease of crops in the South Atlantic and Gulf Coast

States from Maryland to Texas; for this reason it is often referred to as southern bacterial wilt. However, the disease has been reported as far north as Pennsylvania, Ohio, Illinois, West Virginia, and Kentucky.

From the point of view of production, the losses are due to early death of plants, which results in a diminished crop or even no crop. Losses are not restricted to the field, however. Affected tubers may be merely discolored by slight decay of the vascular ring and as a result bring a reduced price, or they may be invaded by secondary organisms and decay completely (p. 5). The disease at one time was reported to be so severe in some fields in Florida that it was necessary to stop growing potatoes, eggplants, and tomatoes in these fields.

Brown rot is sometimes a serious factor on the market in shipments of the early, or new, crop of potatoes from the South, but since practically none go into storage the disease is of little importance as a storage rot.

On the tuber, the external symptoms of brown rot may be a slight depression at the point of attachment or a grayish-brown discolored patch on the surface. Sometimes no external symptoms develop. When infected tubers are cut across at the stem end, a moist, brown discoloration and a slight softening of the water-conducting tissues can be seen (pl. 6, *D–F*). Frequently, though not invariably, gray, sticky masses of bacteria ooze from the discolored vascular area without any application of pressure (pl. 6, *E* and *F*). At this stage there is no odor. Later, as the bacteria work toward the center of the tuber, the entire interior becomes soft and brown and only a thin crust of tissues holds the tuber together. If the rot reaches this advanced stage while the potatoes are in transit, adjacent tubers are likely to become smeared with the rotted contents. In the later stages of the disease, secondary organisms gain entrance, the tissues become creamy in consistency, and a foul odor results.

The symptoms of brown rot are somewhat similar to those of ring rot (p. 37). However, when a diseased tuber is cut the presence of brown rot is indicated if white, sticky droplets of bacteria ooze from the vascular ring and the decayed tissues in the immediate vicinity of the ring are brown to black. Tubers affected with ring rot show no bacterial ooze, although in early stages the soft, decayed tissues can be forced out of the ring area by squeezing the tuber. In the later stages of ring rot, the decay extends into the pith and the cortex of the tuber and the affected tissues are soft or crumbly, and yellow, cream, or reddish-brown discolorations prevail.

In the growing plant the outstanding symptom is wilting, which appears rather suddenly. At first it may be restricted to a single shoot, but it may ultimately affect the entire plant. Sometimes the foliage yellows before it wilts, but if the onset of the disease is sudden, wilting may occur without previous yellowing. The stems shrivel if they are not very woody; usually they turn yellowish green at first and become brown or black finally. A cross section of the stem or leaf petiole shows brown dots or a brown ring, and the discolored vascular tissues often show through the green parts of the stem and petioles as long, dark streaks. Closer examination reveals that the water-conducting vessels are clogged with bacteria, which may ooze out when the stems are cut.

CAUSAL FACTORS

The bacterium that induces brown rot (*Bacterium solanacearum*) occurs in certain virgin soils in the South. This statement is based on observations of the disease in potatoes and other related crops grown from disease-free seed potatoes and on land that had never before produced potatoes. In fact the disease is most severe in crops of potatoes grown on new soil and diminishes in frequency and severity the longer the soils are cultivated or cropped. It has been suggested that some of the conditions which might cause this are (1) increase in the acidity of the soil (brought about either by natural agencies or by the addition of fertilizers), which in turn might reduce the population of *B. solanacearum;* (2) death of the causal bacteria because of the activities of competing organisms in the soil flora; (3) death of the bacteria caused by exposing them to strong sunlight when the field is cultivated; and (4) absence of host plants, which do not ordinarily grow in cultivated fields.

The brown rot bacteria in the soil invade the roots and underground stems of plants and thus gain entrance to the conducting tissues along which they travel or are carried to all parts of the plant. Brown rot seems to progress most rapidly in young plants. If the soil is free of the organism, the latter may be carried into a field by insects from diseased crops in nearby fields. When Colorado potato beetles from diseased plants were allowed to feed on healthy ones, the disease appeared on the latter within 7 to 9 days. About 3 weeks after symptoms appear in the tops, the tubers may be partly or entirely rotted.

Warm, moist weather favors the development and spread of the disease. The organism grows well at temperatures ranging from 77° to 97° F. As the temperature is reduced, the rate of growth is retarded until it is practically stopped at a temperature of 55°.

CONTROL MEASURES

Acidifying the soil by the application of 800 to 1,000 pounds of sulfur per acre in June and then reducing the soil acidity before planting by using 2,000 pounds of limestone per acre has been effective in reducing the incidence of brown rot in Florida. The brown-rot organism does not remain alive in soils with an acidity of pH 4.1.

Differences in resistance to brown rot have been established experimentally for tubers of various potato varieties. The Green Mountain proved to be the most resistant to brown rot and the Katahdin ranked second; it was followed in order by the Spaulding Rose, Irish Cobbler, Triumph, and Chippewa.

Tubers that are known to be diseased should not be stored or shipped even though infection may be only in the initial stages, for such potatoes rot very quickly at temperatures above 55° F. As it is practically impossible to discover and remove all tubers on which brown rot in the early stages occurs, potatoes from affected fields should be used immediately or shipped to nearby consuming centers.

(See *49, 50, 51, 186, 187, 191.*)

793130°—49——2

CHARCOAL ROT

(*Macrophomina phaseoli* (Maub.) Ashby [4])

OCCURRENCE, SYMPTOMS, AND EFFECTS

Although the fungus that causes charcoal rot is present in the soil in the Southern and Southwestern States, it has not been responsible for much decay in potato tubers until recent years.[5] The most serious rot found so far has been in shipments from California, Georgia, Missouri, and Texas. Charcoal rot caused appreciable loss in California potatoes on the Buffalo, N. Y., market in 1941 and on the Chicago, Ill., market in 1944. [6]

Most infections by *Macrophomina phaseoli* occur at the stem end of the tubers; but under some conditions they may occur at the eyes, in enlarged lenticels, and in wounds. Black, moderately firm areas about the stem end of tubers and cloudy blotches about the eyes are characteristic external symptoms of charcoal rot (pl. 8, *A*). The fungus may sometimes invade only the outer tissues but often it progresses throughout the tuber causing a semiwatery, flabby breakdown of the tissues. When diseased tubers are cut the affected tissues appear yellowish to light buff, but soon they change to pinkish buff, brown, and finally black (pl. 8, *B* and *C*). Unlike charcoal rot in sweetpotatoes, beans, corn, and many other crops, that in white potatoes is seldom associated with the small, black sclerotia characteristic of the causal fungus. The decay appears somewhat like leak but the diseased tissues are not so watery, and prominent wound infections common in leak are not usually visible.

CAUSAL FACTORS

Hot, moist soils favor the growth of *Macrophomina phaseoli* and the development of decay; consequently the northern potato crop is likely to remain free of charcoal rot. Experimental evidence indicates that the minimum temperature for growth of the causal organism is near 46° F., the optimum 88°, and the maximum 108°.

Charcoal rot has been reported especially serious in potatoes grown in Texas under irrigation. Maturing plants become infected in the field during hot weather. It is believed that the causal fungus progressing down the stolon into the stem end of the tubers leads to the most extensive decay. When the plants are seriously infected a great many of the tubers rot in the soil, and those harvested during wet, warm weather are very likely to decay during transit or storage. All varieties of potatoes appear to be susceptible to this disease.

CONTROL MEASURES

Well-drained soils planted with early varieties of potatoes that may be harvested before hot weather should produce crops relatively free of charcoal rot. If stem end infections are noted at harvesttime all diseased tubers should be sorted out before shipping or storing.

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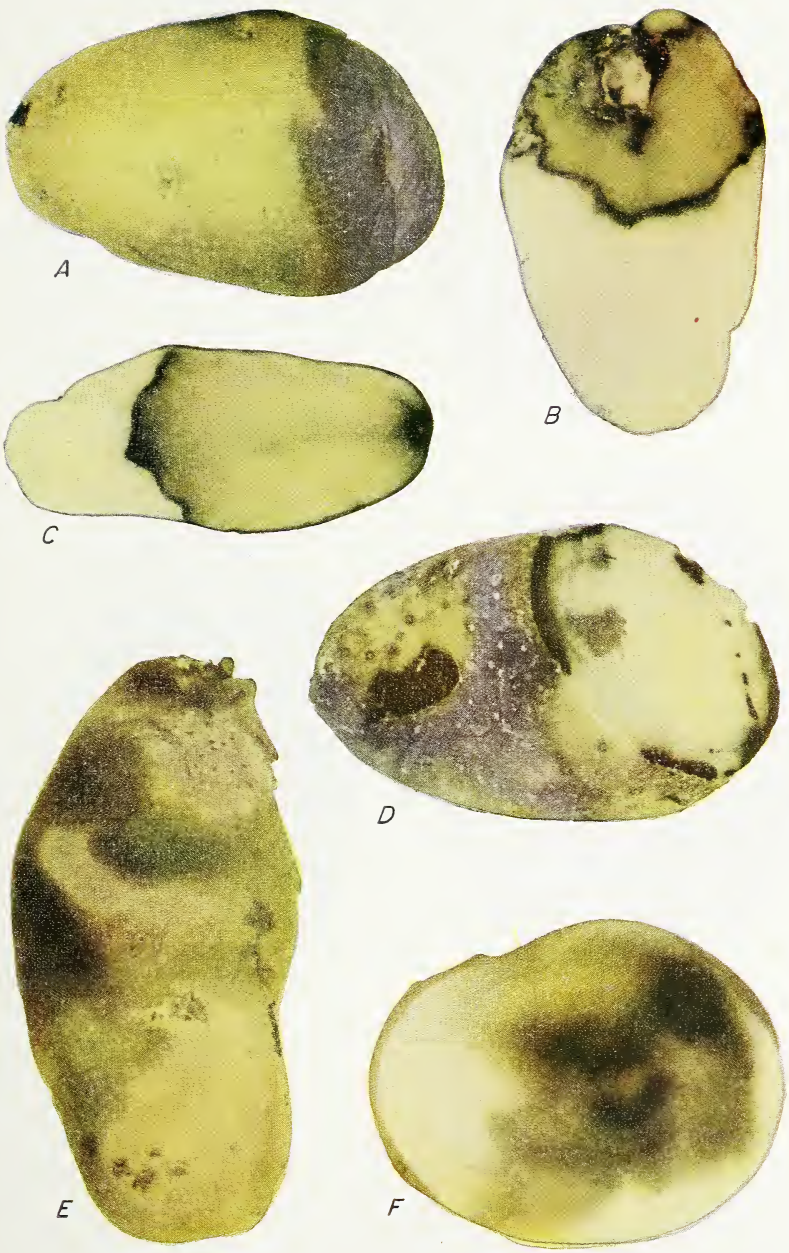
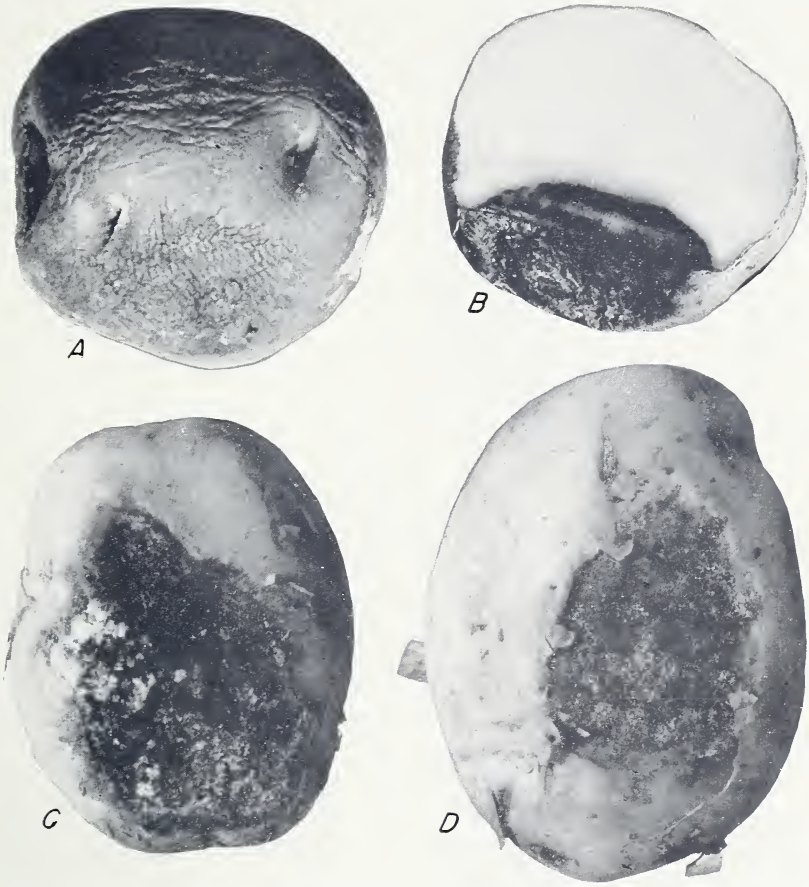
[4] Sclerotial stage, *Sclerotium bataticola* Taub.

[5] Boyd, O. C. A STEM AND TUBER ROT OF POTATO IN GEORGIA. U. S. Bur. Plant Indus., Plant Dis. Rptr. 11: 75. 1927. [Processed.]

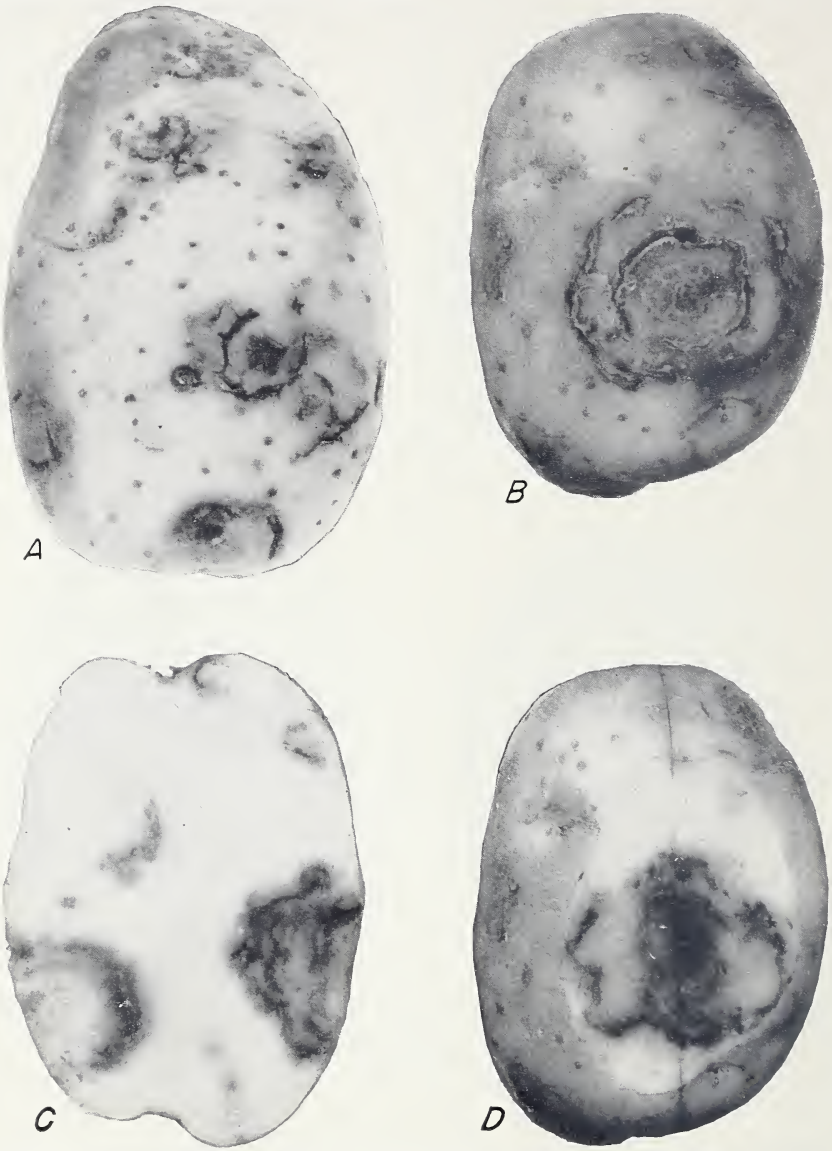
[6] Ramsey, G. B. CHARCOAL ROT IN CALIFORNIA POTATOES. U. S. Bur. Plant Indus., Soils, and Agr. Engin., Plant Dis. Rptr. 28: 758. 1944. [Processed.]



Potato diseases: *A*–*C*, Charcoal rot; *D*, heat injury (scald) in wet soil; *E* and *F*, leak.



A and *B*, Potato tubers showing external (*A*) and internal (*B*) symptoms of chemical injury; *C* and *D*, early potatoes showing transit bruising.



Potato tubers affected with corky ring spot: *A* and *B*, External symptoms; *C* and *D*, internal symptoms.

Since the minimum temperature for growth of the causal fungus is about 46° F., infected tubers held near this temperature during transit or storage should not show much progress of this decay.

(See *202.*)

CHEMICAL INJURY

Chemical injury to potatoes during transit is rare except in shipments made in boxcars that have recently hauled salty hides, fertilizer, or other chemicals. The exterior of the chemically injured areas on potatoes are usually leathery; beneath are flabby, softer tissues extending to a depth of ⅛ to ¼ inch. Sometimes the chemical can be tasted within the killed tissues. When the injury first occurs the tissues are not discolored; but after a short exposure to the air, they become brown to black (pl. 9, *B*) and eventually dry if they are not invaded by decay-producing organisms. Affected tubers are unmarketable.

Potatoes in transit or in storage may be injured by common salt, sodium nitrate, ammonium sulfate, coal tar, and oil products. It has been shown that ground limestone, hydrated lime, and gypsum, which are the chemicals most commonly found in cars, are relatively noninjurious because of their insolubility. Mere contact with these chemicals apparently is not sufficient to induce chemical injury. However, if the substances are ground into the tissue of the tubers by pressure or rubbing, the characteristic symptoms are developed. This occurs when potatoes are in contact with walls or floors that may be impregnated with the chemicals named.

Usually the side of a potato in contact with the chemicals is flattened (pl. 9, *A* and *B*), and the injured tissues are soft and flabby; at times they are semiwatery, very much like those of ordinary pressure bruises. Generally, however, pressure-bruised tissues are not so deep or so dark-colored as those affected by chemicals.

Cars showing chemical residue on the floors and walls should not be loaded with potatoes unless heavy paper or straw liners are used to keep the tubers from contact with the chemicals.

(See *210.*)

CORKY RING SPOT

Corky ring spot has been found only recently in potatoes grown in the United States. In 1946 it was recognized as causing serious damage to the tubers in some fields in Florida. Washington-grown potatoes received on the Chicago market also have shown characteristic symptoms of this disease.[7]

Under the names sprain and internal rust spot this disease has been known as a serious potato trouble for many years in European countries, the Netherlands Indies, and South Africa.

Affected potato tubers show brown rings, semicircles, loops, and spots on the surface (pl. 10, *A* and *B*) and similar brown rings, loops, and spots in the interior tissues (pl. 10, *C* and *D*). The skin over some of the brown, slightly sunken rings often cracks. The surface markings can usually be traced to similar markings within the tuber, but many internal brown spots and semicircles appear to be isolated

[7] Ramsey, G. B., and Smith, M. A. corky ringspot of potatoes from washington. U. S. Bur. Plant Indus., Soils, and Agr. Engin., Plant Dis. Rptr. 31: 8–9. 1947. [Processed.]

from the surface lesions. The affected tissues are moderately firm and corky.

The characteristic ringlike external and internal markings of tubers affected with this disease distinguish it from internal brown spot (p. 25).

The cause of corky ring spot is not known. Until more is known about it, no recommendations for control can be made except the general precaution of making sure that no tubers showing this disease are used for seed.

(See *52*.)

CRACKING

Growth cracks in tubers are generally associated with an uneven water supply and an injudicious use of fertilizers. Growth is checked and then renewed, with resultant cracking of the tough skins. Growth cracks are usually covered with a normal cork layer and therefore are not subject to decay (pl. 11, *A*).

Tuber cracking often is especially serious in Triumph and sometimes it occurs in other varieties at harvesttime (pl. 11, *E*). Sometimes 35 percent of the newly harvested potatoes piled in a bin in a storage cellar show cracking; at other times potatoes seen on the market show considerably more than this. This cracking occurs as a result of mechanical shock in tubers that have developed a high internal pressure from increased water absorption. The amount of mechanical shock necessary to induce cracking may be extremely slight, as, for example, when potatoes are turned out of the soil by a digger. Excessive pressure in tubers results when increased water absorption is accompanied by a sudden decrease in water loss from the leaves. This may result from a drop in temperature, an increase in humidity, or a sudden decrease in leaf area caused by frost or blight damage. Cracking sometimes occurs during the winter when potatoes are removed from a storage cellar for shipment.

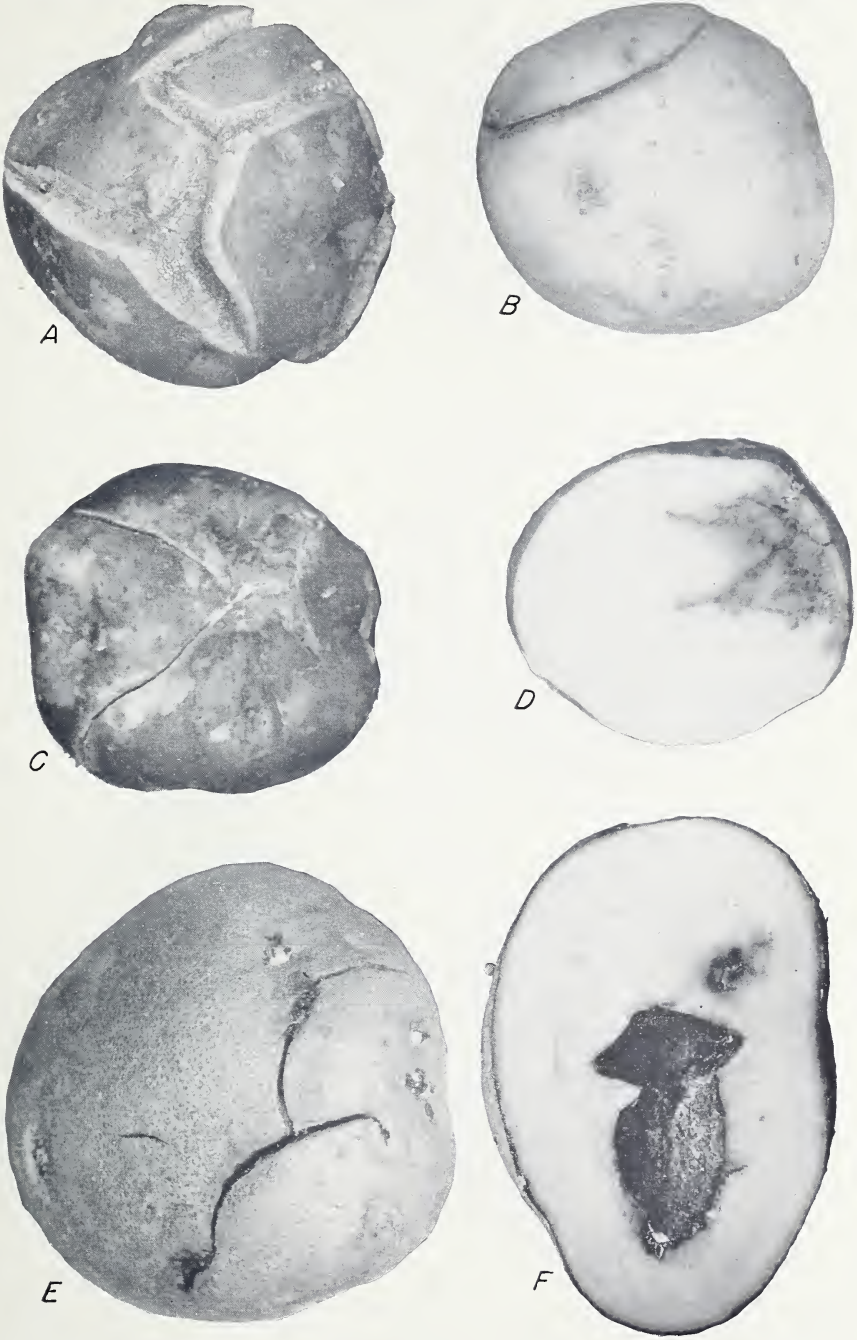
Rot-producing fungi or bacteria may gain easy entrance through freshly made cracks and digger cuts (pl. 11, *B*) and bring about considerable loss in storage. Such loss can be controlled by proper field practices and by providing conditions favorable for wound healing in the storage period. Control of cracking at harvest is obtained by root pruning before digging, by delaying digging when the soil is too wet or the air temperature is too low, correct adjustment and operation of diggers, and care in picking, hauling, and unloading.

Pressure bruises that result in cracks are often noted on tubers taken from heavily loaded cars especially on those next to the floor racks. The cracks may be visible from the outside or may be seen only by cutting into the tuber. They do not extend uniformly through the tuber but present a shattered or netlike pattern in the area on which pressure has been exerted (pl. 11, *C* and *D*; see also pp. 24 and 46). Fungus and bacterial infection of these injured areas may occur if healing does not take place promptly.

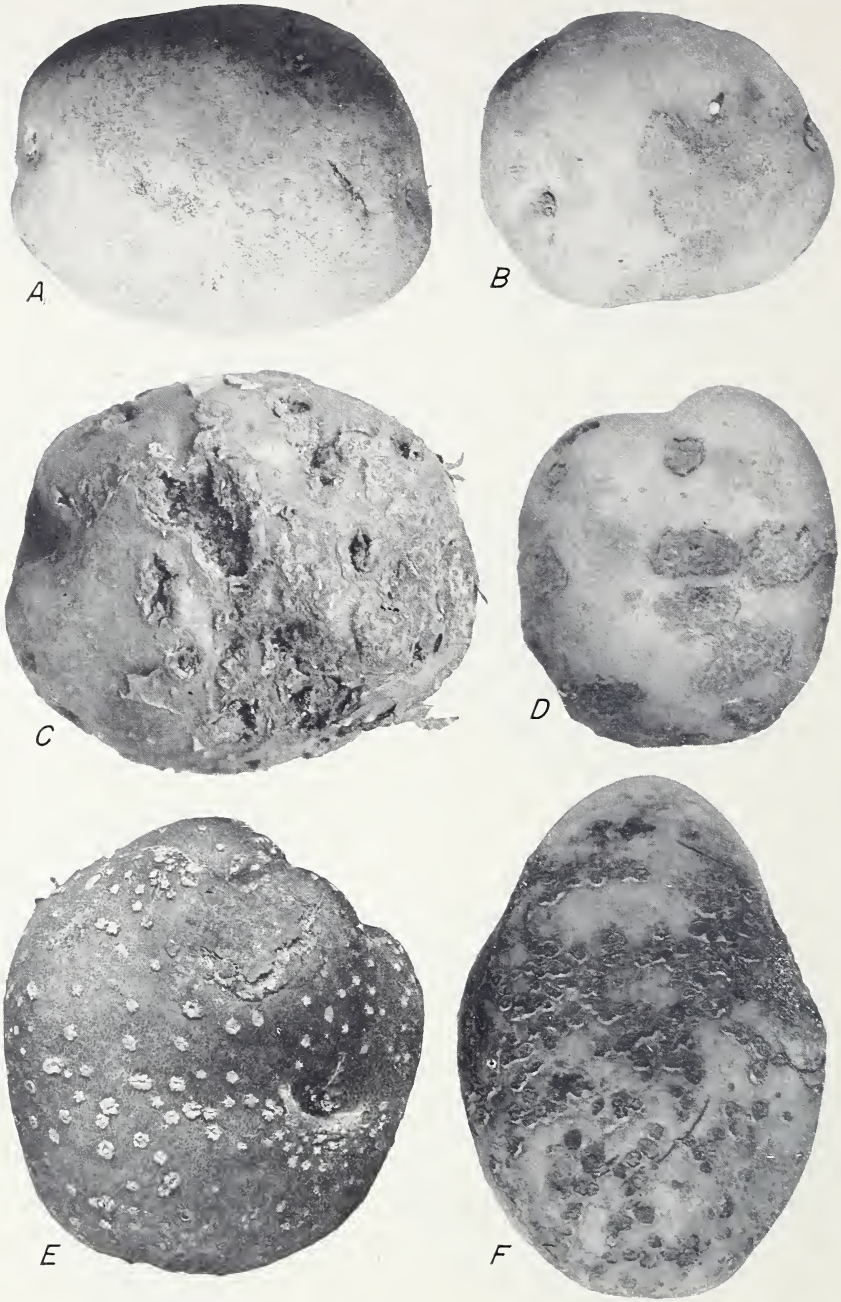
(See *87*, *88*, *123*, *140*.)

ENLARGED LENTICELS

Enlarged lenticels often occur on tubers that are grown in wet, heavy soils. The lenticels on most varieties of potatoes are normally inconspicuous, but under wet growing conditions the early varieties



Potato tubers showing symptoms of various injuries or physiological diseases: *A*, Growth cracks; *B*, digger cut; *C* and *D*, shatter bruises; *E*, surface cracks; *F*, hollow heart.



Potato diseases: *A* and *B*, Silver scurf; *C* and *D*, scab; *E*, enlarged lenticels; *F*, powdery scab.

such as Irish Cobbler, Triumph, and Red Warba are especially likely to develop large protruding lenticels. At first the tissues are whitish and rather soft and are easily rubbed off during washing and handling, thus affording an entrance for decay-producing organisms (p. 5). Enlarged lenticels that are not infected harden and become brown and corky (pl. 12, *E*).

FREEZING INJURY

The varieties of potatoes vary somewhat in their reactions to low temperatures. Some show serious internal discolorations after prolonged storage at temperatures several degrees above their actual freezing point (p. 30); others may not show injury even when exposed to 29° F., the average freezing point of most varieties. It has also been noted that the freezing point of a given variety has a tendency to become lower as the season progresses. Tubers stored for several weeks at low temperatures will stand about 1° lower temperature without freezing than similar tubers that have been stored at 50° or above.

The symptoms of freezing injury (pl. 13) vary with the kind of tissues killed and the amount of tissues involved. Tubers that are frozen solid collapse promptly upon thawing and become soft and watery. Such tubers are often called "leakers." However, the presence of moisture on a tuber is not necessarily a sign of freezing injury, since cold tubers when brought into moist air with a temperature higher than their own soon condense water on their surfaces from the air. When extensive areas of a tuber are killed by freezing, the affected tissues usually become infected with bacteria which cause sticky, slimy, foul-smelling rots under warm, humid conditions. In dry air the frozen tissues may become dried down, tough, and leathery or they may lose moisture and become granular and chalky masses of starch and tissue remnants.

Long exposure of tubers to temperatures just below the freezing point or moderately long exposure to very low temperature usually results in killing of all the tuber tissues; both internal and external symptoms become apparent after thawing. Moderately short exposures just at the freezing point or short exposures to temperatures far below the freezing point usually result in the death of only the most susceptible tissues and external symptoms may not be apparent. When the potato is cut these milder types of freezing injury (freezing necrosis) may show up as slightly dry, gray or bluish-gray patches underneath the skin or as a dark discoloration of the vascular ring, particularly at the stem end. In some instances the internal and external vascular strands (phloem) on either side of the ring (xylem) are also discolored, thus making a netlike pattern in the tuber. In addition to these discolorations dark blue-gray to black blotches in the vascular ring and surrounding tissues are characteristic of one type of freezing injury. In the blotch type of freezing injury that extends to the skin secondary infection by bacteria or species of *Fusarium* occasionally takes place; ordinarily, however, decay rarely follows internal freezing necrosis.

Freezing injury in the field at or before harvesttime can usually be diagnosed by the bluish-gray blotches underneath the skin or the more uniformly soft, flabby, or watery areas on one side of the tuber.

Freezing injury during storage and transit may manifest any or all of the symptoms described; therefore the location of the frozen stock in the storage or in the car gives the most satisfactory clue as to where and when the trouble occurred. Frozen tubers scattered through the load received on the market indicate that freezing occurred in the field or during storage, whereas frozen tubers only along the floor and side walls of the car indicate transit freezing.

Potatoes exposed to temperatures of 40° F. or lower, but not low enough to cause ice formation, gradually accumulate enough sugar to give them a sweet taste. Sweetness of tubers, however, is not a sign of freezing as is sometimes believed. The objectionable sweet taste will largely disappear in potatoes if they are held at about 60° for a week or more. Although potatoes stored between 50° and 60° have better texture, color, and flavor when cooked, it is inadvisable to store them for long in this temperature range because of increased danger of decay and sprouting.

Freezing injury to potatoes may be avoided by protecting them against exposure to temperatures below 29° to 30° F. In very cold weather artificial heating must be resorted to during storage and transit. When this is done adequate vents and air passages must be provided to insure distribution of the warm air; otherwise some of the tubers may freeze and some nearest the source of heat may develop blackheart (p. 7).

(See *92*, *122*, *210*, *211*, *212*.)

FUSARIUM TUBER ROTS

(*Fusarium* spp.)

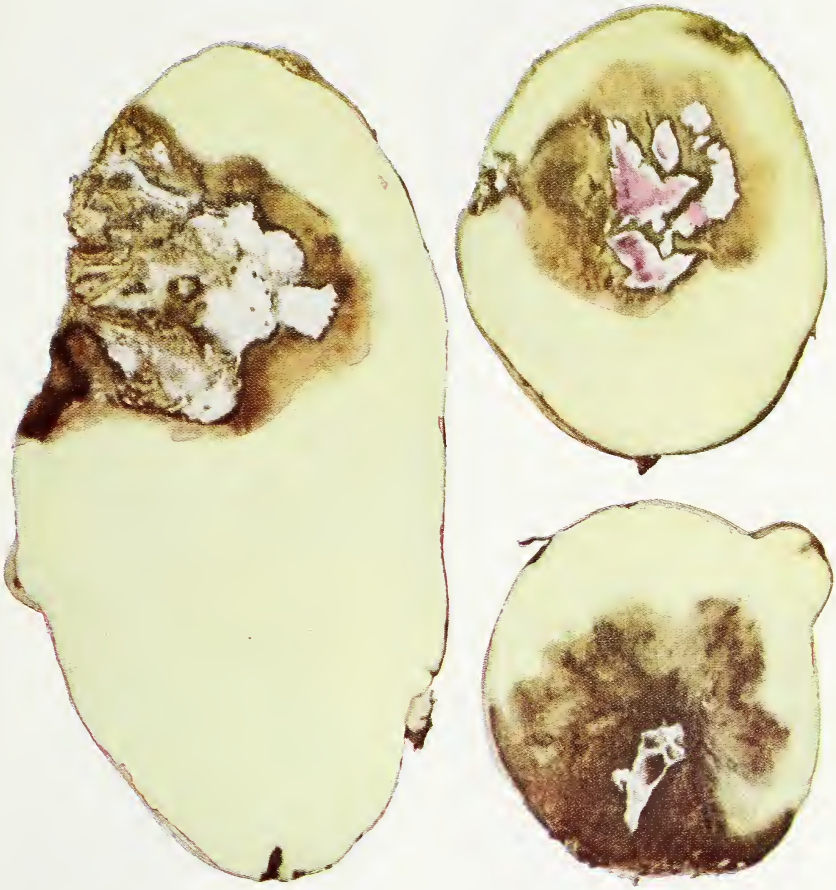
OCCURRENCE, SYMPTOMS, AND EFFECTS

Fusarium rots of various types occur in potatoes from all parts of the United States. They are destructive in tubers primarily during storage and transit, and in late potatoes they usually cause greater losses than does any other disease. Some types of fusarium tuber rot may develop in potatoes in the soil and cause serious decay, particularly at the stem end, but a larger number of infections occur through injuries made during the harvesting and storing of the crop.

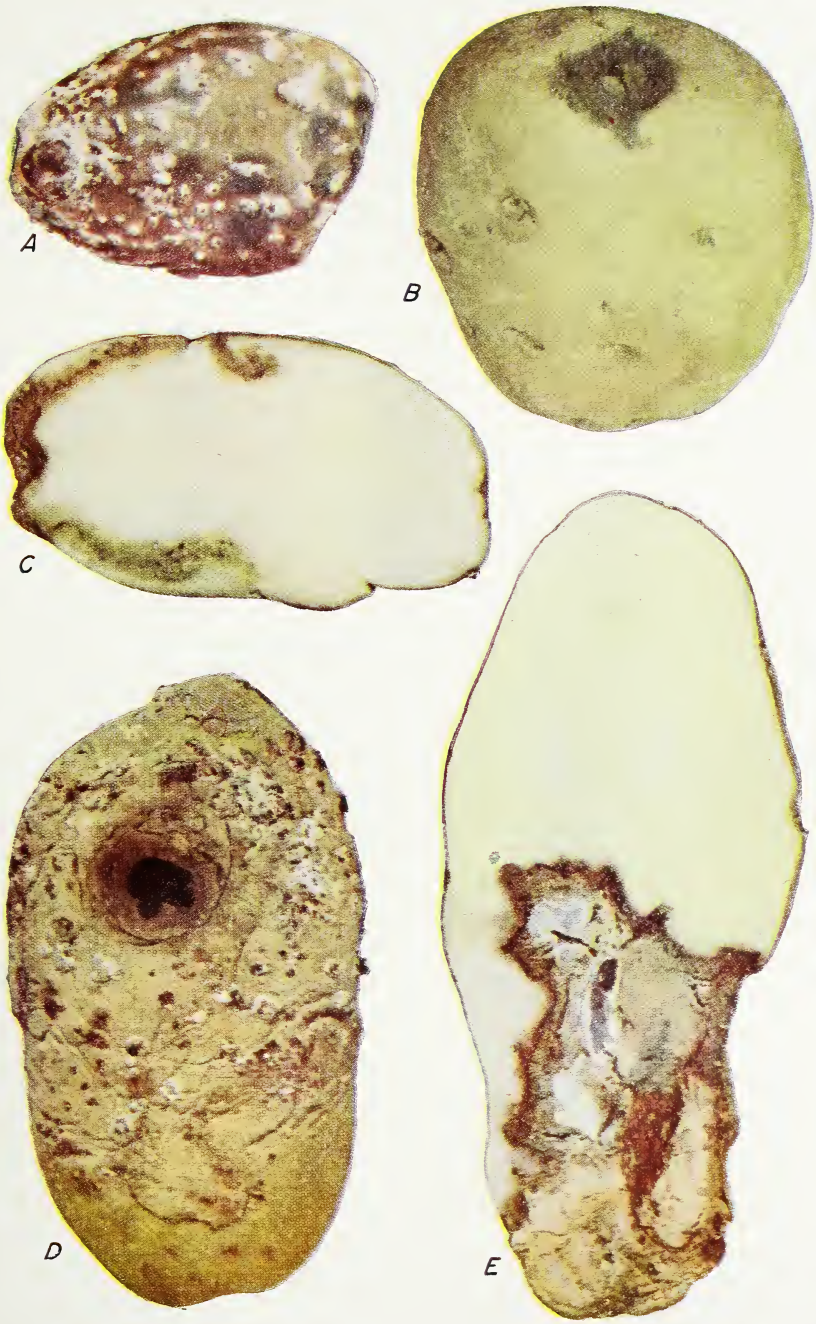
Although the symptoms of fusarium tuber rots (pls. 14 and 15) depend largely upon the species of *Fusarium* causing the infection, the kind of decay is greatly influenced by the temperature and humidity conditions under which the potatoes are held. It is not feasible, therefore, to try to determine the kind of fusarium tuber rot except for technical purposes, and even then cultural and microscopic studies must be made.

As a rule the tuber-rotting organisms enter either through the stem end or through wounds. The color of the affected tissues is light brown at first, becoming darker with age and eventually very dark brown. The decaying tissues soon become sunken, the skin becomes wrinkled, and the small tufts of mold become visible at the eyes, lenticels, injuries, and stem end (pl. 15, *A* and *D*). Species of *Fusarium* are characterized by white, yellow, pink, rose, or bluish-purple molds. At low temperatures the affected tissues are usually dry and firm or even powdery, but at high temperatures they are often moist and pliable.

Potato tubers showing internal symptoms of freezing injury.



Potato tubers affected with wet type of fusarium tuber rot.



Potato tubers affected with dry type of fusarium tuber rot: *A*, *D*, *E*, Advanced stages in storage; *B* and *C*, stem-end infections.

In some types of fusarium rot the causal fungus invades the central part of the tuber, thus breaking down the tissues and eventually causing the formation of cavities that are lined with bright-colored mold (pls. 14 and 15, *E*). In other types moist or dry brown pockets are formed in wounds or at the stem end. When the stem end is invaded (pl. 15, *B*), the fungus grows down the vascular ring, causing it to become discolored; this condition is often accompanied by a glassy or water-soaked break-down of the adjacent tissues (pl. 15, *C*). Externally only a small sunken area at the stem end may be visible, but internal discoloration and decay make the tuber useless.

CAUSAL FACTORS

Many species of *Fusarium* are capable of rotting potato tubers, but the following are of chief importance: *F. trichothecioides* Wr., *F. eumartii* Carpenter, *F. radicicola* Wr., *F. oxysporum* Schlecht., *F. sulphureum* Schlecht., and *F. coeruleum* (Lib.) Sacc. All are soil inhabitants. Several of the species appear to be present in all potato-growing regions, but in a given soil or locality one species is often predominant.

Fusarium radicicola, *F. oxysporum*, *F. eumartii*, and *F. trichothecioides* can in varying degrees attack growing plants in the field and infect the setting tubers. All other species of *Fusarium* are wound parasites and consequently become important only after harvest. Rough handling of potatoes during harvest, storage, or transit often provides wounds through which any of the fungi just listed may enter and produce decay. Mycelium and spores of *Fusarium* are common in the soil, on the tubers, on implements, on containers, and on the floors, walls, and bins of storage cellars.

Whether wound infection takes place depends upon the moisture and temperature conditions, which in turn determine to a great extent the rate of healing (suberization and wound-cork formation) as well as the rate of growth of the decay-producing fungi. At temperatures near 40° F. the healing process proceeds so slowly that little resistance to infection is offered.

Most of the species of *Fusarium* that cause tuber rot grow slowly below 50° F. They are relatively high-temperature organisms that grow best between 78° and 86°. Under most favorable conditions wound infections may begin to show visible decay within 4 or 5 days, and more than half of an average-sized tuber may become involved in 10 to 15 days.

Although infected tubers show little or no progressive decay at storage temperatures near 40° F., the causal organisms are not killed by this temperature or even at temperatures approaching the freezing point. For this reason potatoes having dormant lesions caused by *Fusarium* may begin to show active development of decay whenever they become warm.

CONTROL MEASURES

Wound infections by species of *Fusarium* can be reduced by holding the tubers at moderate temperatures and high humidity for several days after digging, thus providing most favorable conditions for the healing of injured tissues. Since this is not always possible, the safest and surest methods of controlling the rots that are induced by wound

parasites are: Careful handling of tubers to avoid cutting and bruising; sorting out of bruised, broken, cut, diseased, or frozen potatoes; and storing the tubers in well-ventilated places at temperatures between 36° and 40° F.

(See *36, 67, 72, 81, 114, 151, 205.*)

FUSARIUM WILTS

(*Fusarium oxysporum* Schlecht., *F. eumartii* Carpenter, *F. solani* (Mart.) Appel and Wr., and *F. avenaceum* (Fr.) Sacc.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Fusarium wilts occur in the warmer potato-growing regions of the country and elsewhere during warm seasons. These wilts of the potato not only reduce the yield by killing plants prematurely but also cause discoloration of tubers sufficiently to make them unmarketable for table and seed purposes.

Northern potatoes grown under cool conditions sometimes develop a somewhat similar wilt that is caused by *Verticillium* (p. 47).

The tubers produced on plants affected with fusarium wilt may show no external symptoms, but internally they may show varying degrees of brown discoloration in the vascular tissues at the stem end. Mild infections produce so little discoloration that it is often difficult to determine whether tubers show wilt, stem-end browning (p. 45), or vascular browning (p. 46). The most reliable symptom of fusarium wilt is the presence of a definite discoloration of the woody vascular ring (xylem) that can be seen when the tuber is cut crosswise ½ inch or more beneath the point where the stem is attached (pl. 16, *B*). The vascular ring may be discolored partly or entirely. Frequently the water-conducting tissue becomes so hard and woody that the dark-brown discolored strands which extend down into the tuber can readily be removed with a knife point.

A second type of wilt is accompanied by a rot that is characterized by brown sunken areas at the stem end of tubers. Such a rot associated with vascular discoloration usually causes the most extensive type of loss from fusarium wilt. For example, invasion of tubers by one of the most serious pathogens, *Fusarium eumartii*, results not only in a stem-end rot but also in vascular discolorations associated with a degeneration of the adjacent tissues and the formation of a band of light-brown, water-soaked tissues which may extend through the length of the tuber (pl. 15, *B*).

In growing plants *Fusarium oxysporum* and *F. eumartii* induce somewhat different symptoms. The wilt induced by *F. oxysporum* is marked by yellowing around the margins of the lower leaves and finally of the entire top; eventually the leaves wilt completely. The yellowing and wilting usually affect one side of the plant more severely than the other. The first symptom of the wilt produced by *F. eumartii* is a light-green mottling of the leaves accompanied by the irregular bronzing of the upper surface. Under dry conditions the leaves slowly turn yellow; in severe cases the leaves droop, but hang to the stem by a mere thread. During dry weather the wilt progresses rapidly, and the entire plant wilts and dies in a short time. If moisture is abundant, wilting does not result but yellowing, rolling, and rosetting of the top may be accompanied by the development of aerial tubers. In both types the fine roots are destroyed.

CAUSAL FACTORS

Potato wilt is induced generally by *Fusarium oxysporum* and *F. eumartii* and occasionally by *F. solani* and *F. avenaceum*. Like other species of *Fusarium* they are inhabitants of the soil, where they live saprophytically on plant debris. Thus they may attack the plant either from the soil or from infected or contaminated seed pieces. In the case of wilt caused by *F. oxysporum*, root infection from the soil predominates. In the other types the disease originates much more frequently from infected seed pieces. In both cases the fungus grows from the diseased mother plant through the stolons and thus into the new tubers. There is no spread of the disease from tuber to tuber in transit or storage. The killed tissues, however, are subject to invasion by secondary organisms.

The temperature requirements of *Fusarium oxysporum* and *F. eumartii* are somewhat different. To produce the disease the former requires temperatures at least as high as 57° F. with heavy soil inoculation. The most rapid development of the wilt takes place at temperatures above 86° and up to the growth limit of the fungus which is about 95°. The lowest soil temperature at which *F. eumartii* can produce wilt is 52°, and the highest is 86°. The most favorable temperature for infection and development of the rot is from 68° to 77°. Infection occurs in wet and dry soils, starting more readily in wet soil; but the wilt subsequently develops more rapidly in dry soil. Tuber infection, also, takes place more rapidly in wet soils.

CONTROL MEASURES

Wilt infections may originate either from the soil or from infected seed potatoes. Since the pathogen is not necessarily visible in the seed tuber and since not all tubers with vascular discoloration are infected with *Fusarium* species, the surest way to get a clean crop is to plant only certified seed potatoes.

(See *53, 68, 69, 70, 74, 120, 124, 147*.)

GOLDEN NEMATODE

(*Heterodera rostochiensis* Wr.)

The history of the golden nematode in the British Isles and in continental Europe indicates that it is a serious threat to the potato industry in any area where it becomes established. Surveys of the potato-producing sections of the United States have revealed its presence only in relatively localized districts of Nassau County, Long Island, N. Y., where it was reported first in 1941.

The nematodes attack the root system of the potato plant, causing stunting, early dying of the vines, and a reduction in the yield of marketable tubers. Some weeks after the plants are attacked the female nematodes can readily be detected on the roots with the naked eye. They appear first as white, spherical, glistening bodies of pinhead size attached to the roots. Later they turn orange and golden brown in color and readily fall off into the soil. The dead body of the female serves as a protective cyst in which eggs and larvae overwinter in the soil. No symptoms are evident on the tuber itself. The tomato plant is also susceptible to attack.

The golden nematode is spread by any agency responsible for the movement or transfer of infested soil. Thus it may be carried by flowing water, shoes of farm workers, feet of farm animals, farm implements, machinery, bags, baskets, and infested soil clinging to the tubers.

Restrictions have been imposed by the State of New York to prevent the further spread of the nematode and thus to prevent its introduction into other areas. In districts where the nematode has been introduced, control becomes very difficult and at the best is only partially successful. Some measure of control has been obtained by long rotations. There is also evidence that the nematode population may be reduced temporarily by the application of soil fumigants.

(See *37, 43, 118.*)

GRAY MOLD ROT

(*Botrytis cinerea* Fr.)

Gray mold rot is seldom of much importance on potatoes. So far it has been found only in storage potatoes from Maine and California. However, under the proper temperature and humidity conditions it may occur in potatoes from any region.

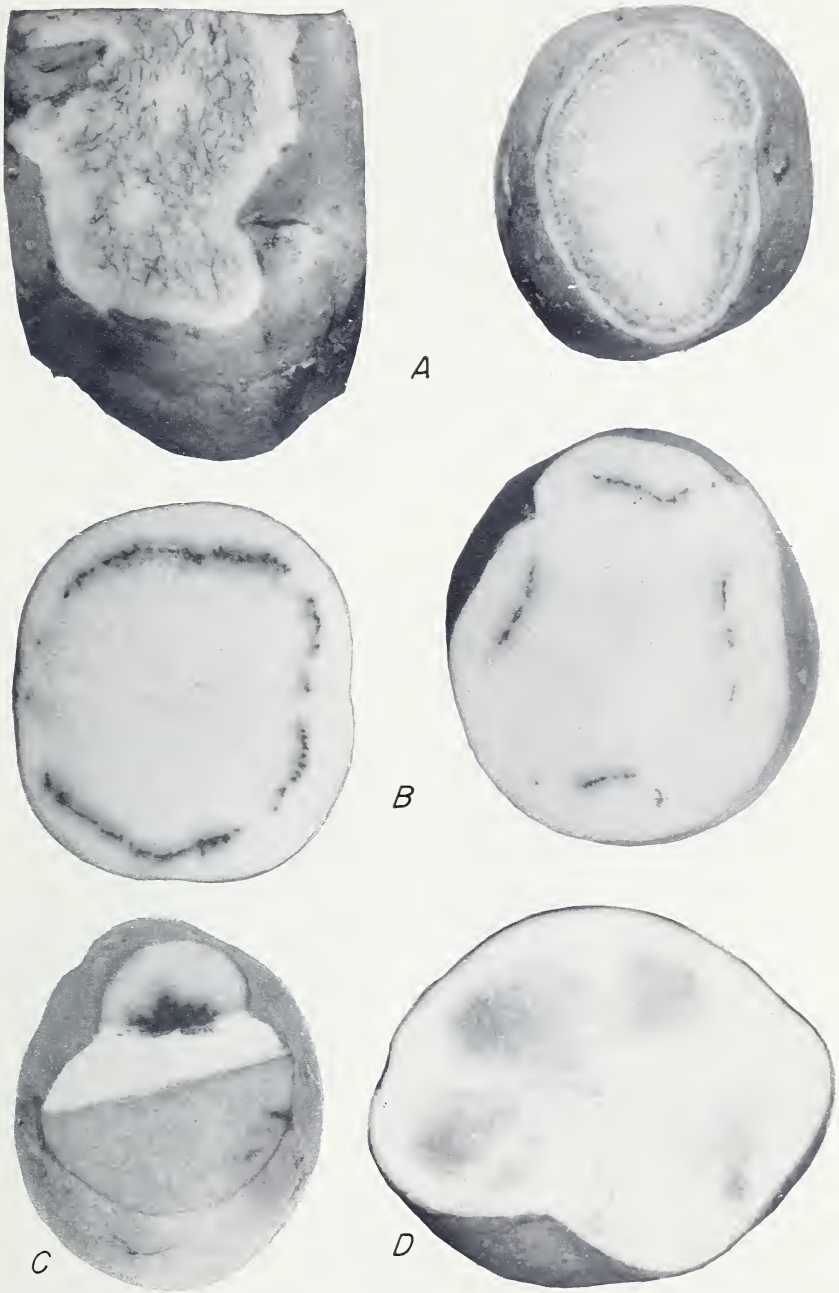
Gray mold rot appears as a pinkish-buff to a brown semiwatery break-down that may extend throughout any or all parts of the tuber. Although infections through the stem end are most common, invasion through mechanical wounds sometimes occurs. In advanced stages of decay the tubers are somewhat withered and flabby with grayish-brown mycelium and granular spore clusters of the causal fungus evident at the stem end, at the eyes, or in mechanical injuries (pl. 17, *F*). After long storage thin, black, irregularly shaped sclerotia may be found in some of the surface mold.

The causal fungus, *Botrytis cinerea,* is widely distributed; occasionally it causes a blight of the foliage and stems of aging potato plants. When this occurs, tuber infection apparently results when the mycelium of the fungus grows down through the stolons into the stem end. Wounded tubers contaminated by spores from diseased vines during harvest may also develop gray mold rot under conditions of high humidity and low temperature.

Experimental wound-inoculation tests have shown that under the usual temperature and humidity conditions at harvesttime in most regions the tubers are able to develop wound periderm soon enough to prevent invasion by *Botrytis cinerea.* For this reason gray mold rot seldom develops in wounds of potatoes held at 70° F., but it does develop readily under high humidity in wounded tubers held at 40°. At this low temperature the formation of wound cork is greatly inhibited, thus allowing the fungus to penetrate the tissues.

Potatoes harvested from plants showing gray mold should be examined for stem-end infections, and all diseased tubers should be discarded. If the disease is suspected, it is advisable to hold the tubers at moderate temperature for 3 days to allow wound periderm to form in the mechanically injured tubers before they are placed in cool or cold storage.

(See *59, 149, 156.*)



Potato diseases: *A*, Net necrosis; *B*, fusarium wilt; C, stem-end browning; *D*, mahogany browning.

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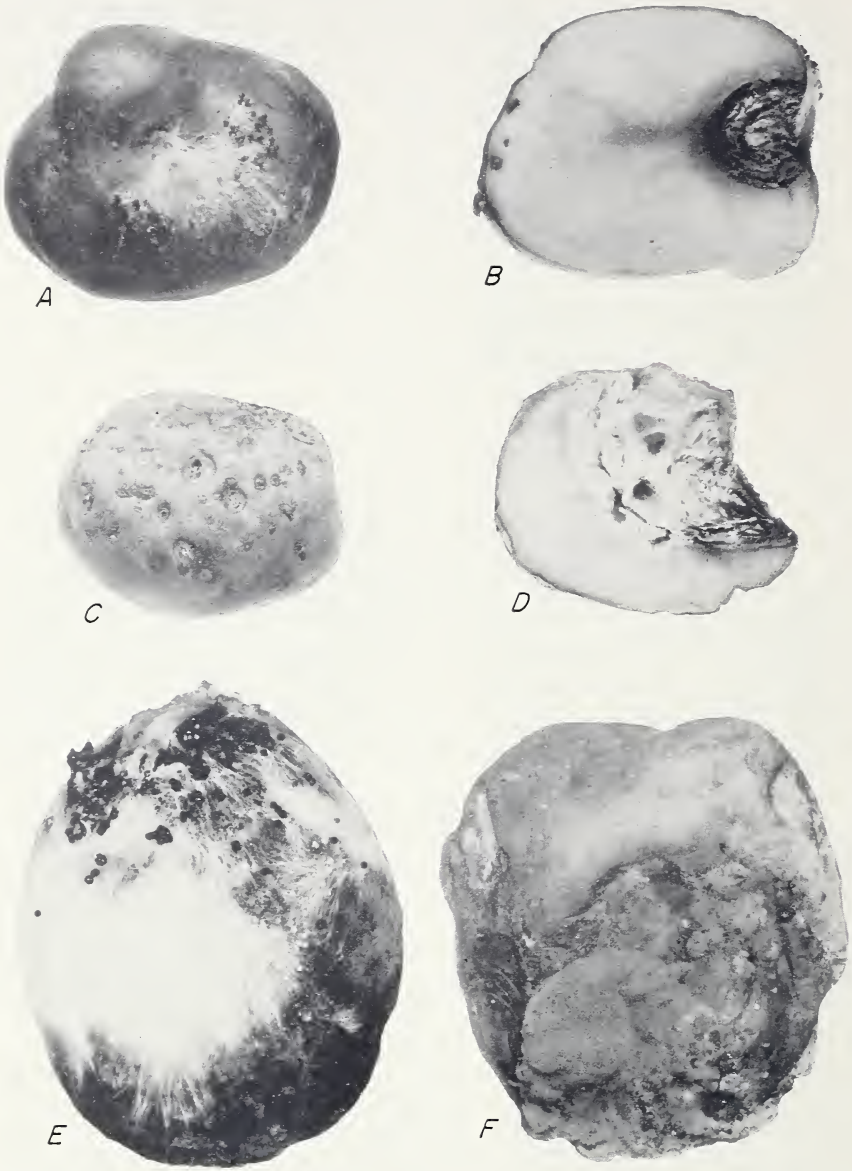


A–E, Potato tubers affected with sclerotium rot: *A*, External symptoms; *B*, internal symptoms on tuber shown in *A*; *C*, symptoms in lenticels; *D*, soft watery stage; *E*, advanced stage showing silky, fanlike mold and sclerotia on exterior. *F*, Potato tuber affected with gray mold rot.

GREENING

Greening is more commonly found in immature tubers than in those fully matured. It is caused by the exposure of tubers to the sun, either in the field or after harvest. Affected tubers often have a bitter taste which renders them unpalatable and possibly dangerous to eat because of the presence of solanin, an alkaloid that is poisonous if taken in sufficient dosage.

The green color is generally restricted to the layers immediately below the skin. If exposure to the light is prolonged, however, chlorophyll develops in the deeper tissues. Once the bitter taste accompanying the green color has developed, it does not disappear completely even though the greened tubers are removed from the light. Affected tissues may also become tough, leathery, and excessively yellow, even without development of chlorophyll.

White varieties of potatoes such as the White Rose and Irish Cobbler are particularly susceptible to greening when exposed to light through open-mesh bags and while on display at retail stores.

HEAT INJURY (SCALD)

Various types of heat injury to localized spots and areas on potato tubers exposed during harvest on hot days are sometimes referred to as scald or sunscald. Although bright sunlight is commonly associated with the excessive temperature that usually injures tubers, it has been found that heat alone will cause this injury. For this reason the term "sunscald" should not be used, while "scald" is a less truly descriptive term than "heat injury."

Heat injury occurs most frequently in the early and intermediate crops of potatoes harvested on hot days from April or May through August. The break-down of tubers due to heat injury and the subsequent decay in the injured tissues are the most important factors in causing loss in shipments of these potatoes. A great majority of the potatoes injured by heat in the field become infected by soft rot bacteria (p. 5). An extensive development of this decay, usually referred to as slimy soft rot, is one of the best evidences that market potatoes were excessively warm at some period in the harvesting or marketing process.

In severe cases of heat injury the exposed surfaces of tubers soon become watery and blistered, but injuries of less degree may or may not show external signs of tissue break-down for several hours. With exposure to dry air the injured tissues not infected by bacteria lose moisture and become sunken, flat, leathery areas that often appear somewhat like field freezing injury. When first cut through heat-injured tissues may appear white to gray, but after exposure to air they become brown and finally black as in blackheart (p. 7).

It is not possible to judge by air temperature alone whether potatoes will be injured by heat during harvest; for on a moderately hot day heat accumulated by tubers at their exposed surfaces may cause death to the tissues. The surface tissues of exposed tubers will often show temperatures between 20° and 30° F. higher than the air temperature. Temperatures as high as 135° have been recorded on the sunny side of tubers left exposed in the field on a hot day. The thermal death point of potato tissue is believed to be near 122°.

At temperatures below this point potato tissues are not likely to break down because of death by heat alone. Such temperatures are, however, definitely dangerous to freshly dug potatoes because they are affected physiologically in such a way as to predispose them to attack by soft rot bacteria.

In some localities the term "scald spot" has been used to describe oval or irregular, sunken, brown areas that occur on skinned parts of tubers that have been exposed to dry wind. This injury is not a true scald, since it is not dependent upon high temperatures but is a type of desiccation injury related to browning (p. 9).

During excessively hot weather in wet seasons tubers at or near the surface of the soil may become so hot that internal as well as external injury results. This "deep scald" (pl. 8, *D*) appears as a grayish to purplish watery break-down of the internal tissues sometimes with purplish-black discolorations showing at and under the skin. In waterlogged soil the tubers are somewhat smothered by the free moisture; when free moisture is combined with high temperature blackheart (p. 7) may develop.

In order to protect potatoes against heat injury at harvesttime it is necessary in many regions to pick up the tubers within 15 to 30 minutes after they are dug. Such precaution is always advisable when the air temperature is above 90° F. After they are picked up, the potatoes should be protected from intense heat by bags or a tarpaulin while they are standing in the field and on loads. This not only gives protection from heat injury but also prevents excessive discoloration of bruised and skinned areas on tubers when hot winds or low-humidity conditions prevail (p. 9). In some regions it may be advisable to dig the potatoes late in the afternoon, to allow them to cool overnight, and to pick them up early the following morning.

Since potato tubers may be injured by heat without showing external evidence of break-down of tissues for several hours, caution should be exercised in shipping potatoes suspected of having been thus injured. If it is possible to hold the potatoes a day before grading and loading, a great deal of decay in transit can be avoided. Shipping the potatoes under refrigeration can also be relied upon to minimize possible losses from the bacterial soft rot that always follows as a secondary effect.

(See *143*, *144*, *146*, *157*, *165*, *167*.)

HOLLOW HEART

Hollow heart is a condition brought about by too rapid or too irregular growth. It often occurs during wet seasons in potatoes grown in very fertile or heavily irrigated soils. At times it seriously affects the marketability of potatoes.

Hollow heart consists of more or less irregular cavities of varying size in the center of the tuber (pl. 11, *F*). The cavity is usually lined with light-brown to brown dead tissue. Hollow heart does not develop in transit or in storage. Decay seldom follows it except when tubers are cracked enough to allow bacteria and other soil organisms such as *Fusarium* spp. to enter.

Since hollow heart is often more prevalent in large tubers, regulation of the rate of growth may be desirable. It has been reported

that reduction in size of tubers can be obtained by planting whole tubers or seed pieces close together. These measures, however, may not prove practicable.

(See *137, 207.*)

INSECT INJURIES

There are a number of insects that attack potato tubers and cause various types of injury. The descriptions herein cover the most important insect injuries found on market potatoes.

(See *121, 134.*)

FLEA BEETLE INJURY

The larvae of the tuber flea beetle (*Epitrix tuberis* Gent.) and to a less extent those of the potato flea beetle (*E. cucumeris* (Harr.)) and of the western potato flea beetle (*E. subcrinita* (Lec.)) feed on potato tubers and cause injury (pl. 18, *A*). In feeding on or near the surface of young tubers they produce pimples and channels in and under the skin. Deeper feeding results in the formation of brown splinterlike pegs extending ⅛ to ¼ inch into the tuber. These corky splinters, pimples, and channels are so tough and dry that usually no secondary infections by bacteria or fungi occur.

Injury by the tuber flea beetle is severe in eastern Colorado, western Nebraska, Oregon, and Washington. The western potato flea beetle causes little damage to the tubers, although it is associated with the tuber flea beetle. The potato flea beetle has a wide range, but in the Eastern States, where it is most abundant, it does not usually cause much damage to tubers.

SPOTTED CUCUMBER BEETLE INJURY

In the early producing areas in the South the larvae of the spotted cucumber beetle (*Diabrotica undecimpunctata howardi* Barber) sometimes tunnel into potato tubers, making surface holes (pl. 19, *A*). When the tubers are invaded while they are small, the remaining channel is rather deep by the time the tubers are of marketable size. In red-skinned varieties of potatoes the lining of these channels and pockets is red (pl. 19, *B*).

TUBERWORM INJURY

The potato tuberworm (*Gnorimoschema operculella* (Zell.)) does its greatest damage to the tubers after they have been dug. Injury, however, may occur in exposed tubers before digging. The larvae migrate from the stems and leaves to exposed tubers. Harvested tubers left in the field overnight may also become heavily infested by larvae moving from vines to tubers. After the eggs hatch on the tubers either in the field or in bins or sacks, the larvae at first work under the skin and later tunnel through the flesh (pl. 18, *D* and *E*). The entrance is often made at the eyes, where sawdustlike heaps of material is evidence of the activities of the tuberworm. After transit or storage the infested tubers are often found riddled with deeply penetrating channels that make them unmarketable (pl. 18, *D*).

The damage by this insect is more severe in certain sections of California and the Southern States in years of low rainfall.

WIREWORM INJURY

Wireworm injury is caused by the larvae of the snapping, or click, beetles (Elateridae). This injury is of several types. The larvae burrow into the growing tubers, the depth and size of the burrow depending on their size and length. Large worms attacking tubers late in the season bore clean-cut, round holes rather deep into the tuber (pl. 19, *C* and *D*). These channels and burrows may be brown and lined with cork or may become plugged and surrounded by dead tissue caused by secondary infection with *Rhizoctonia*.

Holes made in tubers by quackgrass and nutgrass sometimes appear similar to those made by wireworms. However, the remains of these grasses in the tuber usually serve to identify this kind of injury.

INTERNAL BLACK SPOT

Internal black spot, or black spot, (pl. 19, *E* and *H*) is a tuber discoloration that for a number of years has been a serious problem on Long Island. Tubers from other growing areas have occasionally been found with similar symptoms.

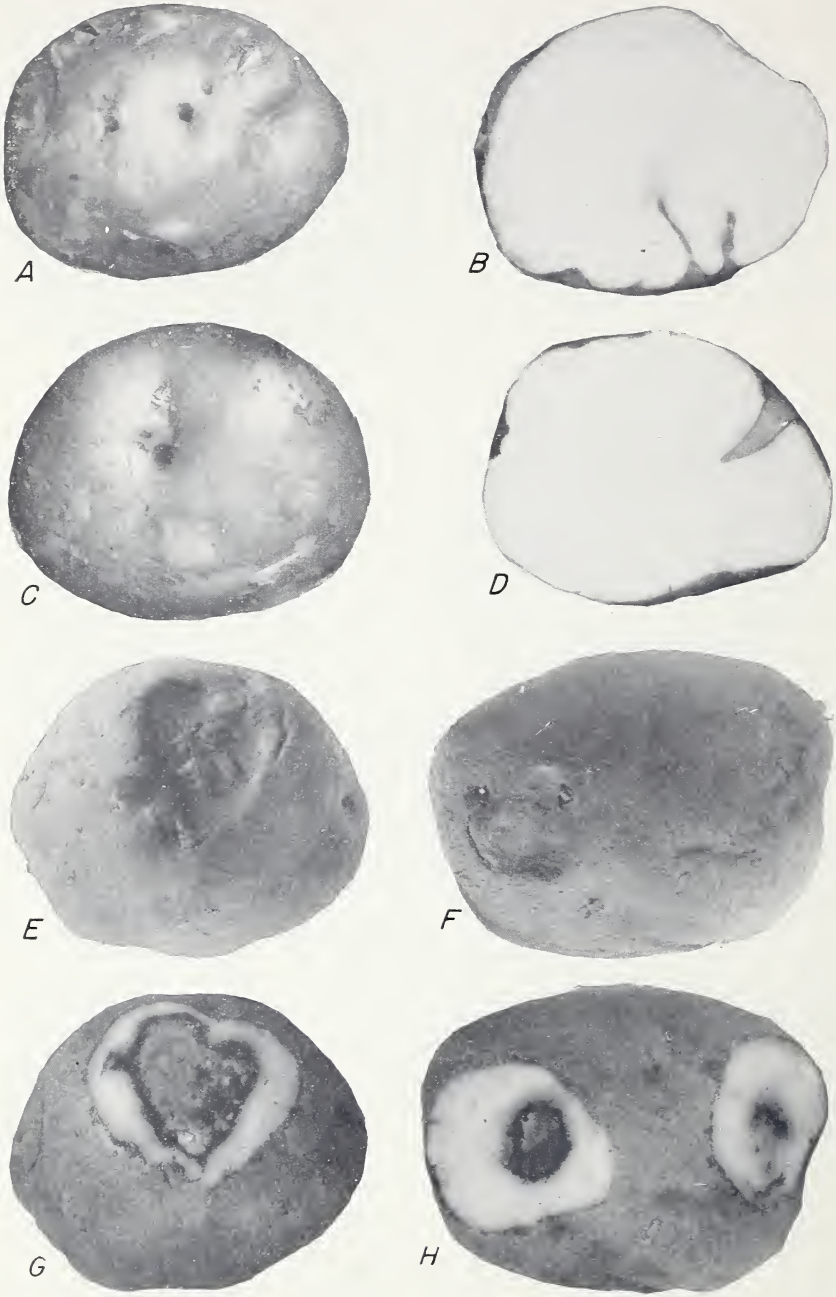
As indicated by the name, the chief symptom of internal black spot is a blackening of the internal tissues (pl. 19, *G* and *H*). Typically the affected regions are confined to the cortex (tissues lying between the skin and the vascular ring); occasionally the tissues somewhat deeper are affected. In some instances the discoloration does not extend to the surface so that there are no external symptoms to indicate the presence of black spot, but when a thin tangential slice is pared from the tuber the blackening becomes readily apparent. As thus viewed the spots are variable in outline but tend to be roughly circular or oval; the borders of the darkened tissues are somewhat irregular, and although fairly definite they are not always sharply delimited. The spots vary greatly in size.

In more severe cases of black spot external symptoms are strikingly evident (pl. 19, *E* and *F*). This is brought about by a rapid drying out of the blackened tissues and of the thin layer of overlying normal ones. As a consequence the surface of the tuber over the black spots becomes irregularly sunken to a depth of 1/16 to 1/8 inch.

At first the blackened tissues are of nearly normal texture; later they dry out and become starchy. Eventually they become sharply delimited through the formation of a thin boundary of cork tissue. Ordinarily there are, in the affected tissues, no splits or cracks such as are found in shatter bruises. Black spot may appear in potatoes within a day or so after harvest. Ordinarily, however, most of the black spot becomes evident after the tubers have been in storage for several months.

Investigations have shown that black spot is a form of bruising injury caused by the bumps and falls that occur when the potatoes are shoveled up, dumped into the grader hopper, and otherwise handled during the grading and sacking operations. When black spot occurs shortly after harvest, it is the result of bruising during digging. Black spot differs from ordinary mechanical injury in that (1) the injury is internal without either skin rupture or the formation of a shatter bruise; (2) it may be produced by very slight applications of force; and (3), as already pointed out, the affected tissues become blackened.

Potato injuries: *A*, Flea beetle injury; *B* and *C*, root knot (nematode injury) *D* and *E*, tuberworm injury.



A–D, Potato tubers showing different insect injuries: *A* and *B*, Cucumber beetle injury; *C* and *D*, wireworm injury. *E–H*, Potato tubers affected with internal black spot. Note that *E* and *G* are the same tuber and *F* and *H* are the same tuber.

Symptoms of black spot thus may not be apparent even in the bin or during grading. However, black spot can readily be detected 24 hours after the time of injury—in fact the maximum discoloration will already have occurred by that time. When external symptoms appear it may be several days before all the pronounced indentation of the tuber surface takes place.

Observations made in farm storage cellars have shown a close relation between the occurrence of pressure bruises on the tubers and the subsequent appearance of black spot after grading. The pressure bruises, or pressure spots as they might more appropriately be called, are somewhat softened, flattened, or slightly indented areas where adjacent tubers have been pressed together by the weight of the potatoes above them in the bin. The pressure bruises of themselves have little commercial importance because they tend eventually to round out and become inconspicuous after the potatoes have been removed from the bin. However, when the pressure-bruised spots are hit or bumped during grading and sacking they readily develop black spot. In fact a very high percentage of black spot, particularly the more severe type, originates at pressure bruises. It should be pointed out, however, that black spot may appear anywhere over the surface of the tuber.

The factors which make certain lots of potatoes susceptible to either pressure bruises or black spot are unknown.

Where internal black spot is an important factor every effort should be made to handle the potatoes as carefully as possible in preparing them for market.

(See *208*.)

INTERNAL BROWN SPOT

Internal brown spot has been noted on the market in potatoes from a number of regions. During certain seasons it occurs commonly on Long Island, where it was particularly prevalent in the 1944 and 1948 crops. The tuber discoloration as observed on Long Island [8] appears to be identical with that which occurs on certain sandy, gravelly soils of Wisconsin and which has been under study there for a number of years. A somewhat similar trouble with or without brown discoloration of the vascular tissue sometimes occurs in California and Idaho potatoes where, because of its association with high soil temperature, it is called heat necrosis.

The symptoms of internal brown spot [9] are confined to the tuber and are not apparent externally. Affected potatoes are found upon cutting (pl. 20, *C*) to have light- to reddish-brown regions of dead tissue scattered promiscuously throughout the flesh of the tuber. Frequently the discoloration is found chiefly at the bud end of the tuber. The spots may vary in number from few to many and in size from mere specks to large masses ½ inch in diameter. The borders of the spots are irregular and rather indefinite in outline. Affected tissues are firm or somewhat corky. In some instances the

[8] Observations made by J. S. Wiant both on the market and at growers' storage cellars throughout the marketing period of the 1944 crop and during the 1948 harvest season.

[9] The more general term "internal necrosis" has been applied to the tuber discoloration in Wisconsin.

discoloration may resemble that found in tubers affected with yellow dwarf (pl. 20, *E*).

Internal brown spot develops in the tubers before they are dug. Although it has been shown to increase in amount and severity as the season progresses, there is no further increase after the tubers are removed from the soil. The cause of the trouble is unknown, but it appears to be physiologic. The occurrence of the disease has been correlated with high temperatures and low humidities—in other words, it has been of most importance during hot, dry seasons. Just what particular combination of temperature and moisture conditions favor its development has not been determined.

Tuber discolorations somewhat similar in appearance to internal brown spot have been reported from other countries under such names as rust spot, internal rust spot, sprain (p. 13), and internal brown fleck. Various causes have been indicated for them.

(See *105*.)

JELLY-END ROT

Jelly-end rot occurs chiefly in Burbank and Russet Burbank potatoes grown in California and Idaho. The tubers that have long, pointed ends and are succulent and deficient in starch are especially susceptible to invasion by species of *Fusarium* and *Rhizoctonia*, which cause a soft, jellylike type of decay (pp. 16 and 35). At first the rot produced is soft, light brown, and somewhat translucent, but with loss of moisture it becomes brown to black and moderately firm (pl. 7, *A* and *B*). This type of decay may be found at harvest-time as well as during storage.

No satisfactory control measures are known.

(See *36*, *180*.)

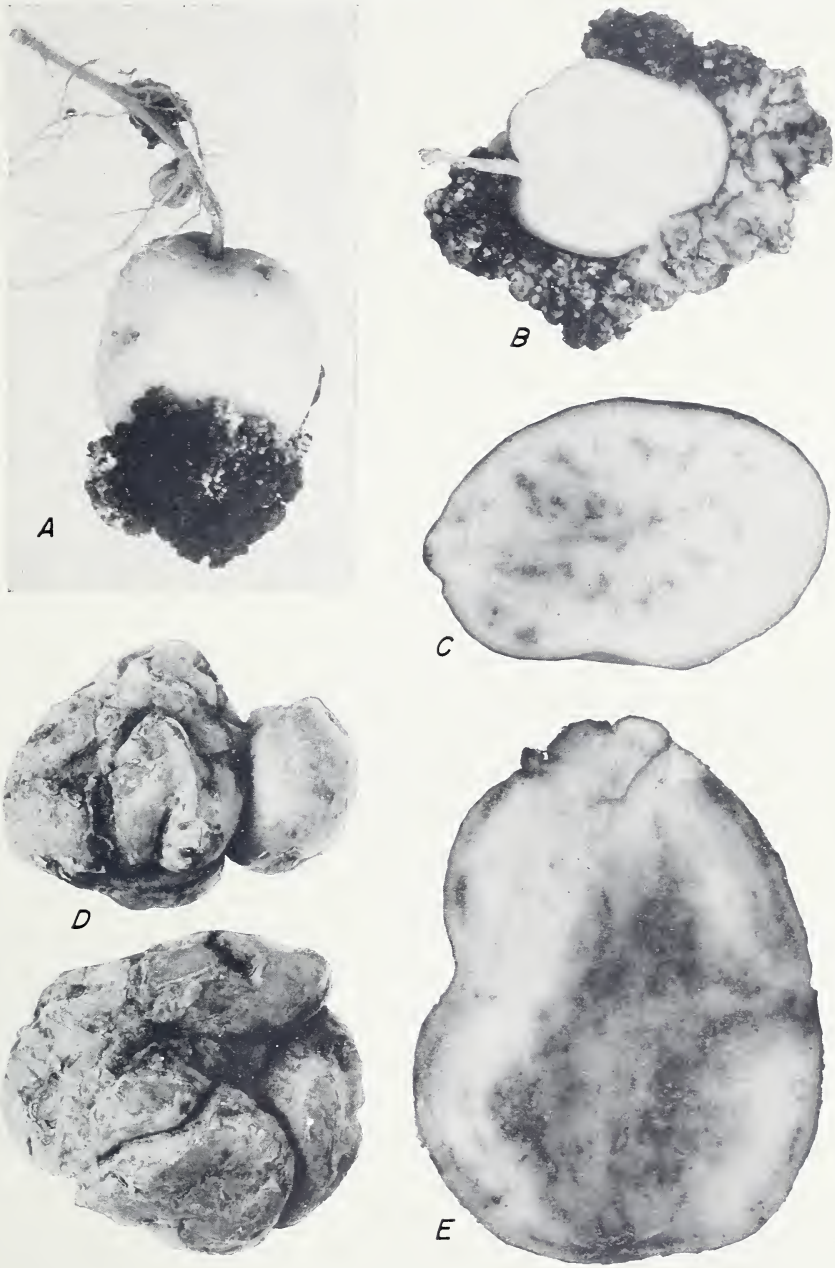
LATE BLIGHT

(*Phytophthora infestans* (Mont.) DBy.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Late blight is one of the most important diseases of potatoes. During wet, cool seasons it may be responsible for serious crop losses in any potato-growing region. It is commonly found important in early potatoes in the Atlantic and Gulf Coast States. Occasionally it seriously damages the early potatoes in the Rio Grande Valley of Texas and in some potato-growing sections of the Pacific Coast States. Usually, however, the most serious outbreaks of this disease occur in the late-potato-producing States extending westward from the New England States through the Middle Atlantic States to and including the States bordering on the Great Lakes.

On the leaves and other above-ground parts of the plant late blight appears as water-soaked or pale-greenish spots that soon turn brown or nearly black. As the spots enlarge and darken, the advancing edges of the lesions retain their water-soaked appearance. A characteristic sign of the disease is the sparse whitish growth of mold that may be seen at the border of the lesions on the under side of the leaflets. Under dry conditions the affected tissues become dry and brittle; under humid conditions they become soft and decayed and have a strong odor. The disease may spread throughout the plant



Potato diseases: *A* and *B*, Wart; *C*, internal brown spot; *D* and *E*, yellow dwarf.

and completely kill the foliage. When the disease becomes serious it is commonly found that all plants in a given area of the field have been killed.

On the tubers late blight appears as reddish-brown, brown, or purplish-brown areas, which, as development continues, become shrunken and darker colored (pl. 21, *A*, *E*, and *F*). In storage stock late blight tuber rot is moderately dry, firm, or leathery, and it does not usually extend more than ⅛ to ¼ of an inch beneath the skin (pl. 21, *D* and *G*); but in newly harvested potatoes, particularly those of the early southern crop, the reddish-brown moist decay often penetrates to a depth of ½ inch or more (pl. 21, *B* and *C*). The decay may also occur in the tissues at the stem end as a broad brown ring, and in advanced stages the tuber may be converted into a shriveled mass. In newly harvested stock or in stock held in very moist conditions, the white tufts of the causal fungus often appear at the eyes, breaks in the skin, and the lenticels.

A serious feature of the disease on tubers is that it opens the way for bacteria and for other fungi which rot the tuber much more rapidly than the late blight fungus. If bacteria get in through diseased spots, a very wet, soft, or slimy and foul-smelling rot results (p. 5). This usually occurs under warm, moist conditions. If other fungi (usually species of *Fusarium*) follow it, there results an odorless rot which may be wet or dry, depending upon temperature and moisture conditions (p. 16).

CAUSAL FACTORS

Late blight is caused by the fungus *Phytophthora infestans*, one of the downy mildews. The fungus lives over from crop to crop as mycelium in diseased tubers. When such tubers are planted, it grows up the sprouts and produces the disease on the emerging stalks. Here under moist conditions the fungus bears spores which may be carried by splashing rain, by wind, and possibly by insects to neighboring healthy plants. In this manner late blight may become established (particularly on the lower, shaded leaves) in many fields, but if conditions are not favorable for extensive spread of the disease, it may remain rather inconspicuous. Of equal or possibly even greater importance in establishing early primary field infections are the diseased tubers that are thrown out as culls around packing houses or are discarded on dump piles in the vicinity of potato fields; under such conditions the fungus likewise develops on the surface of partially exposed tubers and on vines growing from infected tubers. These volunteer plants often bear great numbers of spores which may be carried by winds to potato fields.

Once late blight becomes established in scattered areas in a field, the occurrence of certain sequences of moisture and temperature conditions brings about the rapid spread of the disease. Detailed studies have proved that the fungus will produce sporangia abundantly at a temperature range of 64° to 77° F. and a relative humidity of nearly 100 percent, provided those conditions prevail for at least 6 hours. A lower temperature range (54° to 59°) and a relative humidity of 100 percent prevailing for 12 hours likewise provide favorable conditions for sporangial formation.

If exposed to dry air the sporangia soon become unable to germinate; however, if they are held in the presence of moisture for 30 minutes or longer at temperatures of 50° to 59° F., they germinate and produce large numbers of swarm spores, which in turn soon infect healthy leaves. A duplication of these conditions gives rise to another crop of spores. Frequent repetition of this cycle soon spreads the disease to wide areas of the field while wind-borne spread of sporangia sets up similar diseased areas in other fields, thus giving rise to an epiphytotic of the disease.

From diseased vines the spores either fall or are washed to and into the ground by rains; consequently, tubers which are borne within an inch or two of the surface or which are not completely covered with soil may become contaminated with the spores of the fungus. Tubers will also become thoroughly covered with spores if they are dug while diseased vines are still green or if they are piled in the field and covered with diseased vines.

The presence of spores on a tuber, however, does not necessarily mean that the tuber will become infected. Under either dry conditions or temperatures below 40° F. or above 86° the fungus will not grow. On the other hand, since some time must elapse between penetration of a tuber by the fungus and the appearance of the first symptoms, it frequently happens that tubers which look sound but in which the fungus has established itself are stored or loaded for shipment. Such infected tubers will develop symptoms only if temperature and moisture conditions are favorable. The fungus may lie dormant in tubers for months and then begin to grow as soon as favorable temperature conditions arise. In some experiments it was found that inoculated tubers did not develop the first symptoms for 3 weeks at 64° to 71° in moist air. Apparently sound tubers taken from a field where the tops were killed by the fungus were stored at 40°, 55°, and 70°; they showed 17, 53, and 19 percent, respectively, of rotting tubers at the end of 2 months. In moist air with temperatures above 41° the fungus grows out of diseased tubers and produces spores that can infect nearby tubers through the unbroken skins. It has been found that under storage conditions, in the presence of moisture and with temperatures between 60° and 71°, lesions on tubers may enlarge at the rate of 2 inches in 10 days.

CONTROL MEASURES

Although it is not possible to exclude entirely the pathogen of late blight from a field, there are two control measures that may do much toward eliminating or reducing many of the early field infections: The first is the use of seed that is as free as possible of late blight infections; the second is to exercise care in disposing of cull potatoes.

When only small lots are involved, discarded tubers, which usually include some that are infected with late blight, can readily be disposed of by feeding to livestock, burning, or burying. If it becomes necessary to establish dump piles, rigorous measures should be taken either to prevent sprout formation or to kill sprouts and stalks as they develop. Some growers do this by covering the dump piles with straw which they then burn. The use of any of several herbicides is effective. In all cases, however, early and frequent inspections of cull piles should be made so that treatments may be properly timed

Potato tubers affected with late blight rot. Symptoms on late potatoes: *A* and *F*, External; *D* and *G*, internal. Symptoms on early potatoes: *E*, External; *B* and *C*, internal.

or repeated as necessary. Dumping of cull potatoes aimlessly over the farm area where they might be overlooked or forgotten should be avoided.

Spraying the vines with bordeaux mixture is the chief measure for control of late blight. Time and details of application vary with the season and the locality; therefore local authorities should be consulted. Copper dusts, although possibly not as effective as bordeaux mixture, may be more conveniently applied under certain conditions. Investigations of a number of other and newer fungicides are under way, but so far none can be unqualifiedly recommended as preferable to bordeaux mixture.

When late blight is prevalent in a field, harvest must not begin until the infected tops have been killed by frost or a herbicide or have died from natural maturity if tuber infection is to be prevented. Tubers with any evidence of decay should be discarded during harvesting and storage operations. Ideal storages are dry and well-ventilated and are held as close as possible to a temperature of 36° to 40° F.

The use of the somewhat blight-resistant variety Sebago will reduce losses from late blight. A number of new varieties that are immune or very highly resistant have been developed and should soon be commercially available.

(See *25, 26, 30, 41, 90, 133, 136, 141, 142, 159, 160, 161, 194, 195.*)

LEAK

(*Pythium* spp.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Although leak has been reported from many States, it has been of chief importance in the potato crops in Idaho and California, where during certain seasons it has been responsible for serious losses both at harvest and during storage. At such times it is also an important market decay.

Symptoms of leak are variable. The first evidence of decay is usually around wounds or in the vicinity of the stolon attachment. Here the skin shows a brownish, somewhat water-soaked discoloration. The flesh may be of normal turgidity or slightly depressed. The boundaries of the enlarging lesions are frequently sharply defined both externally and internally by a dark-brown line, but this is not necessarily so. The most characteristic symptom, however, is the extremely watery nature of the affected tissues. The water is held by the tissues until pressure is applied, whereupon the skin is broken and a yellowish or brownish liquid is readily released. It is this characteristic that justifies the name leak. When the decay is found in stored potatoes, large quantities of juice from rotten tubers drip down on the pile.

Leak frequently affects a large part of the tuber (pl. 8, *E* and *F*). Under some conditions the inside of the tuber may be extensively rotted, even when only small external lesions are visible. Only a thin outer layer, or shell, ¼ inch thick may remain sound. The term "shell rot" has at times been used to designate this condition; but this term, as generally used by growers and dealers, probably includes more than the decay under discussion.

Internal tissues affected with leak are grayish to brownish. On exposure to air they soon turn darker brown or reddish and finally black.

CAUSAL FACTORS

Leak is caused by *Pythium debaryanum* Hesse, by *P. ultimum* Trow, and probably also by other species of *Pythium*. These are common soil-inhabiting fungi that cause damping-off of a wide variety of crop-plant seedlings and rots of a number of different vegetables. One of them (*P. debaryanum*) has also been reported causing a wilting of growing potato vines accompanied by a rotting of the tubers in the field. These fungi are particularly destructive under unusually wet conditions.

Infection of tubers usually takes place through cuts and wounds. At times the fungi attack the stolon and from there enter the tuber at the stem end. Hot weather and abundant moisture favor infection and development of leak. When infection has taken place the rot progresses very rapidly at the optimum temperature range (70° to 85° F.). Entire tubers may then be rotted in only a few days but development is slow between 40° and 50°.

Leak may develop in transit or storage in tubers which are infected in the field during digging but show no visible break of the skin and appear to be free of the disease. There is little evidence, however, of spread from diseased to sound tubers during storage or transit.

CONTROL MEASURES

When leak threatens to be of importance special consideration should be given to keeping the tubers as cool and dry as possible during harvesting and loading as well as in transit and storage. Since skin breaks facilitate infection, all possible care should be taken during harvest to protect the tubers from mechanical injury of all kinds.

(See *18, 28, 75, 82, 93.*)

MAHOGANY BROWNING

Mahogany browning, or internal mahogany browning, is a form of low-temperature injury that affects Chippewa and Katahdin tubers. The chief symptom, as indicated by the name, is an internal reddish-brown discoloration of the tuber flesh. This discoloration is usually found in irregular patches that may occur anywhere in the tuber. The color of affected tissues varies in intensity and may shade gradually into that of normal flesh (pl. 16, *D*). Thus the boundaries of affected areas are irregular and indefinite and there are no sharp lines of separation between discolored and normal tissue. At times, particularly when only the outer tissues are affected, the reddish-brown areas resemble somewhat those of late blight. However, the tissues affected with mahogany browning are not decayed but are of normal firmness and consistency. Affected tubers are undesirable for eating.

Mahogany browning develops when tubers of susceptible varieties are exposed for long periods (about 20 weeks or longer) to a temperature near 32° F. Chippewa and Katahdin are the most susceptible varieties. Mahogany browning is in no way connected with freezing injury

because it develops at temperatures well above the freezing point. Usually it first appears in the northern-stored crops some time during March. It occurs in those parts of the bins where the potatoes have been unusually cold. It has been reported that Chippewa tubers infected by the leaf roll virus tend to show more severe mahogany browning in the outer tissues (cortex) than elsewhere, whereas after long storage at 32° tubers not infected by the leaf roll virus develop mahogany browning chiefly in the vascular ring and pith. Green Mountain and Sebago tubers, although not susceptible to mahogany browning, may develop a blackish discoloration of the flesh when stored for 20 weeks at 30° to 32°.

Mahogany browning can be prevented by storing Chippewa and Katahdin potatoes at temperatures of 38° F. or possibly slightly lower. (See *84, 126, 128.*)

NET NECROSIS (LEAF ROLL)

OCCURRENCE, SYMPTOMS, AND EFFECTS

The term "net necrosis" as used in this publication refers only to that particular kind of necrosis caused by current-season infection of certain varieties such as the Green Mountain by the leaf roll virus. The action of the virus results in a killing of the conducting tissues through which food is transported into and through the tuber. The internal discoloration thus produced is sometimes a serious market factor.

Although the food-conducting channels, or phloem strands, are distributed throughout the entire tuber, net necrosis occurs most frequently at the stem end; however, visible symptoms may extend into the tuber to various depths. The affected strands become hardened, turn dark brown or blackish, and are very conspicuous in the cut tuber (pl. 16, *A*). Their appearance varies with the angle at which the tuber is cut. Thus in cross sections they may show up as dots or streaks. The netted appearance from which the name is derived is best seen by paring the tuber lengthwise in such a manner as to remove only a thin layer of the outer flesh.

When the discoloration does not penetrate far into the tuber from the stolon attachment, early stages of net necrosis are frequently difficult to distinguish from advanced stages of stem-end browning (p. 45). Usually, however, stem-end browning penetrates no deeper than about ¼ to ½ inch, the affected strands are darker and more nearly continuous than in net necrosis, and seen in cross section a tuber affected with stem-end browning usually shows fewer concentric zones of discoloration than does one affected with net necrosis. On tubers symptoms of fusarium wilt and of the mild type of discoloration known as vascular browning (p. 46) are readily distinguished from those of net necrosis by the fact that they are largely confined to the water-conducting tissues (xylem), which in a cross section of the tuber show up as the vascular ring. At times freezing injury produces in tubers discolored strands that very closely resemble those of net necrosis. Usually, however, the strands are more blue gray and not as distinct or as sharply defined as those of net necrosis; furthermore, in most instances a given lot of tubers showing the net type of frost necrosis will also show other types of freezing injury (p. 15).

793130°—49——4

CAUSAL FACTORS

When tubers affected with net necrosis are used for seed, the virus moves up into the growing plant and produces the disease known as leaf roll. Affected plants are stunted, have a more upright aspect, and as indicated by the name show a pronounced upward rolling of the leaves. Tubers of such plants are small and frequently on germinating produce only spindling sprouts; they do not, however, develop net necrosis although they carry the leaf roll virus.

Leaf roll is spread in the field almost entirely by aphids which transmit the virus from diseased to healthy plants during feeding. Under conditions favorable for its spread the build-up of virus within a given seed stock may be very extensive during even a single season.

Spread of the virus usually is greatest late in the growing season. Current-season infections ordinarily do not produce leaf roll symptoms in the plants, but they are solely responsible for the production of net necrosis symptoms in the tubers. However, many tubers that carry the virus as a result of current-season infection may never develop net necrosis. Studies at the Maine Agricultural Experiment Station have shown that there is a definite relation between storage temperature and the development of net necrosis in infected tubers. Maximum development occurs at about 50° F. The peak of the curve for seasonal development of net necrosis at this optimum temperature is reached about 90 days after digging. It has been found that tubers of the Green Mountain variety infected with the leaf roll virus will be largely prevented from subsequently developing net necrosis if promptly after digging they are conditioned by holding them for 60 days at 70° or for 30 to 60 days at 32° to 36°. Thereafter there will be little development of discolored strands regardless of the subsequent storage temperature. Net necrosis may increase during transit if the changes in temperature are toward the optimum for its development.

CONTROL MEASURES

Control of net necrosis involves primarily the reduction of the spread in the field of current-season leaf roll. This in turn requires control of the insect carriers and the use of seed relatively free of the virus. Both are difficult to accomplish. Various control measures recommended are the carrying out of a foundation-seed program, the use of insecticides, destruction of the weed hosts of the aphids, and early harvesting of tops. Progress has been made in developing resistant varieties. A number of new seedlings already developed are known to be highly resistant. Chippewa and Katahdin, although susceptible to leaf roll, remain practically free of net necrosis. Wherever there appears to have been extensive current-season spread of the virus in susceptible varieties such as Green Mountain, reduction in losses from net necrosis can be obtained by conditioning the newly harvested tubers by holding them at effective temperatures as previously explained. Unfortunately, however, this is not ordinarily practicable.

(See *58, 60, 62, 63, 64, 65, 127, 128, 129, 130, 179.*)

PINK ROT

(*Phytophthora erythroseptica* Pethyb.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Pink rot is an important tuber rot of the potato in several European countries. There the disease is found chiefly in wet, poorly drained soils in which the pathogen lives over from year to year and attacks the growing plant through the stolon. In the United States the disease has been reported in small amount from several States on a few occasions, and during 1943 and 1944 it was found frequently in the Idaho crop.[10] So far it has been found only a few times on the market.

The decay typically starts at the stem end of the tuber. The skin over the affected areas is brown and darkening occurs at the lenticels. Diseased tissues are somewhat soft, pliable, and rather wet. When the tubers are cut, the decayed tissues are nearly normal in color but with exposure to air soon turn first pink, later dark reddish brown, and eventually black. Similar blackening is also evident at the lenticels and at cuts or other skin breaks.

In Idaho, pink rot was found frequently to affect the outer tissues of the tuber, which then could be readily sloughed off to leave a central core of sound tissue. Another characteristic was the ease with which the skin could be slipped off from affected areas of the tuber. In Idaho the term "water rot" has been used for the decay, but it appears likely that as used commercially this term includes not only pink rot but also other tuber troubles.

CAUSAL FACTORS

Pink rot is caused by *Phytophthora erythroseptica*, a fungus closely related to the late blight pathogen. Artificial inoculations have shown that several species of *Phytophthora* other than *P. erythroseptica* will produce typical symptoms of pink rot. A species which appears to be identical with or similar to *P. erythroseptica* has been obtained from potato tubers shipped to the Chicago market [11] from Missouri (pl. 1, *C* and *D*), Nebraska (pl. 1, *E–G*), and Colorado. At times the symptoms are somewhat like those of late blight tuber rot (p. 26); in storage stock the decay was frequently of a wet, leaky nature.

Temperature studies have shown that *Phytophthora erythroseptica* makes most rapid growth at 75° F. and causes most rapid tuber rot at 77°. The minimum temperature for development of the pathogen is between 39° and 46°, and the maximum temperature is below 93°.

CONTROL MEASURES

Control measures in Europe include change of seed, crop rotation, and good soil drainage. So little is known about the disease in this country, however, that specific control measures cannot be recommended.

(See *34*, *35*, *47*, *87*, *94*, *101*, *148*.)

[10] BLODGETT, E. C. WATER ROT OF POTATOES. U. S. Bur. Plant Indus., Soils, and Agr. Engin., Plant Dis. Rptr. 29: 124–126. 1945. [Processed.]

[11] Unpublished laboratory notes by G. B. Ramsey.

POTATO ROT NEMATODE

(*Ditylenchus destructor* Thorne)

The only known occurrence of the potato rot nematode in the United States is in Idaho, where it was first noted in 1943 and where it apparently is confined to a limited number of fields in a single locality.[12] It was found in Prince Edward Island, Canada, in 1945. During the early part of 1946 seed potatoes shipped from there to Long Island were found infected with the nematode but so far as is known none of the infected seed potatoes were planted.

Only tubers are attacked. The presence of small gray or brown spots of decaying tissue at the tuber surface is the first symptom of the trouble. Affected tissues dry out as the lesions enlarge and the skin shrinks and cracks. In more advanced stages the decaying tissues form a shallow, dark-brown layer just beneath the epidermis. Tissues for some distance deeper are bluish gray. The injury may be serious in the field at the time of digging, and it may increase in severity during storage.

The nematodes are abundant in the decaying tissues and move from there into the soil. Because they do not form cysts, they should be less difficult to control than either the root knot nematode (p. 39) or the golden nematode (p. 19).

The chief control measures applicable to the potato rot nematode in this country are the prevention of its spread or if possible the eradication of it in Idaho and the prevention of its introduction elsewhere by infected seed potatoes.

(See *6*, *43*.)

POWDERY SCAB

(*Spongospora subterranea* (Wallr.) Lagh.)

Powdery scab is not widely distributed in the potato-growing regions of the United States, but it has been reported from time to time in restricted localities in some of the Northern States where moist, cool conditions prevail. It is rarely found affecting potatoes on the market.

The chief symptom of powdery scab is the presence of raised pustules on the surface of the tuber. These may occur singly or in groups anywhere on the tuber (pl. 12, *F*). At first the lesions are covered by the tuber skin; later this breaks or is partly worn away. The open pustules at this stage are filled with a brown more or less powdery mass of spore balls and characteristically are bounded by the fringed, curled-back remnants of the tuber skin. The spore balls are readily dispersed, thus leaving craterlike depressions bounded by the curled-back skin. The pustules of powdery scab are more nearly circular, more definite in outline, and on the average smaller than the lesions of common scab. As in common scab no soft rot is produced and only occasionally do secondary organisms like *Fusarium* follow in the powdery lesions. However, severely affected tubers are likely to wither in storage more rapidly than normal ones.

[12] BLODGETT, E. C. THE DISTRIBUTION OF PARASITIC NEMATODES ON ECONOMIC PLANTS IN IDAHO. U. S. Bur. Plant Indus., Soils, and Agr. Engin., Plant Dis. Rptr. 30: 182–190. 1946. [Processed.]

Powdery scab is caused by *Spongospora subterranea*, a fungus which may overwinter either in the soil or on diseased tubers. There is no spread of the disease from tuber to tuber in transit or storage. In the field the disease is favored by wet soil and cool weather.

Seed-potato treatments are ineffective as control measures. It is advisable not to plant infected tubers in areas favorable for development of the disease.

(See *103, 138, 155.*)

RHIZOCTONIA BLACK SCURF

(*Pellicularia filamentosa* (Pat.) Rogers)

OCCURRENCE, SYMPTOMS, AND EFFECTS

The most prevalent manifestation of *Rhizoctonia* infection of potato tubers is the so-called black scurf. This consists of hard, brown to black masses of the fungus, known as sclerotia, which adhere firmly to the skin of the tuber (pl. 22, *B*). They vary greatly in size and shape and are composed of an extremely close weft of fungus threads which serve to carry the fungus over winter. They are harmless to the tubers and unless they are present in large numbers do not injure their sale for eating.

A type of injury sometimes ascribed to *Rhizoctonia* is known as russet scab. (See also Russeting, p. 41, and Scab, p. 41.) This blemish occurs when the fungus irritates the superficial layers of skin and causes the development of slightly scabby russeted patches that sometimes cover the whole tuber (pl. 22, *C*). When *Rhizoctonia* is associated with or preceded by insect attack, severe injury to the tuber results. After flea beetle injuries *Rhizoctonia* is likely to produce deep cracks and furrows by penetrating the insect channels and punctures. It also often invades the holes made by wireworms and deepens the pits and channels.

Rhizoctonia seldom induces decay in potato tubers, but at times it is reported as causing a dry, punky decay at the stem end; together with bacteria and other fungi, it causes a jelly-end rot in the succulent pointed ends of Burbank and Russett Burbank potatoes (p. 26). Under some conditions it also invades lenticels and produces small, dry pits, or corelike pockets, that can be lifted from the tuber with the point of a knife.

In attacking the stolons, roots, and main stem of potato plants, *Rhizoctonia* causes sharply sunken brown lesions on the cortex that often are so extensive they girdle the stem or root. If these lesions develop at or near the tender growing tip they frequently destroy it completely. Successively developed sprouts may be destroyed in a similar fashion, so that a cluster, or rosette, of sprouts without growing tips is formed. If this continues long enough, either very weak spindling shoots or none at all come through the ground and poor stands result. If shoots and stolons are attacked less severely or later in their life the plant is not killed, but there is so much interference with the movement of food and with other functions that the leaves roll up, wilt, and become discolored. Often small tubers are developed in the axils of the leaves and stems, or the nodes become excessively swollen because of the interrupted movement of food from the leaves to the underground parts.

Under very moist, warm conditions in the field, the fungus may develop its spore stage at the base of plants, just at or slightly above the ground level. The spore-bearing mycelium consists of an ashen-gray network that forms a film around the stem. It is so friable that it rubs off readily, and with age it cracks and falls off. The presence of this collar of mold is generally not observed.

CAUSAL FACTORS

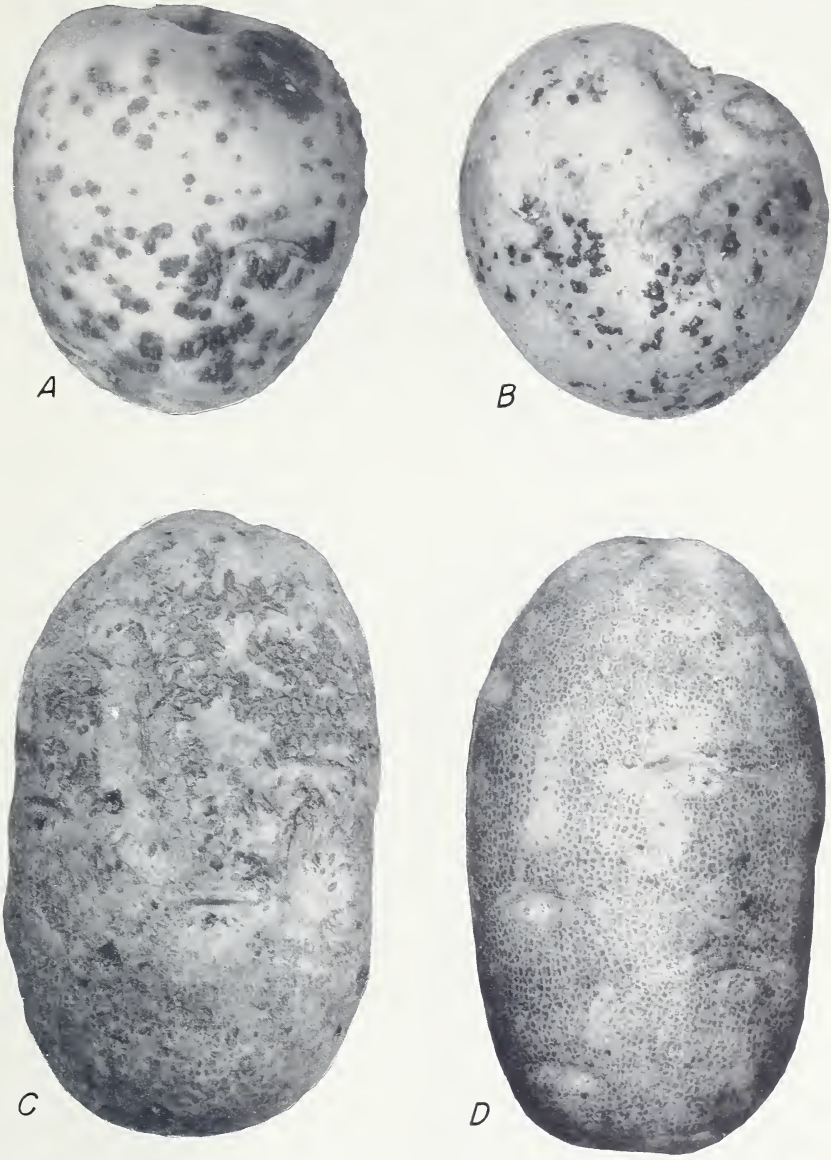
The fruiting stage (*Pellicularia filamentosa*) occurs far less frequently than the sterile stage (*Rhizoctonia solani* Kuehn). The fungus occurs in most soils, living on decaying plant trash and cultivated plants. The likelihood of getting stem lesions and of cutting off sprouts and roots is greater when untreated tubers bearing sclerotia are planted than when sclerotium-free tubers or disinfected tubers bearing sclerotia are used. This indicates that not only the fungus in the soil is responsible for the disease, but probably even more so the fungus that arises from germination of the sclerotia on seed tubers. The spores play a minor role in the propagation and distribution of the fungus; however, there is some evidence that strains of the fungus from spores are more strongly pathogenic than those from sclerotia.

Field observations and experimental work indicate that both soil moisture and temperature determine the occurrence and pathogenicity of *Rhizoctonia*. When the water content of the soil is moderate (19 to 40 percent), infection appears to be determined by several factors. One is the presence of virulent strains. There seem to be a great number of strains of *Rhizoctonia*, many of which are not highly pathogenic. Given a virulent strain and a susceptible host, the temperature of the soil appears to be the most important limiting factor for the development of the fungus as well as for the appearance and severity of the disease. Field observations indicate that temperatures below 70° F. are more favorable for the development of the fungus than higher ones, although in pure culture optimum growth is made at 77°. Frequently the stems and roots of plants that show few or no sclerotia during the summer become literally covered with them at the onset of cooler weather. The disease is more severe in cool seasons and sections than in warmer ones.

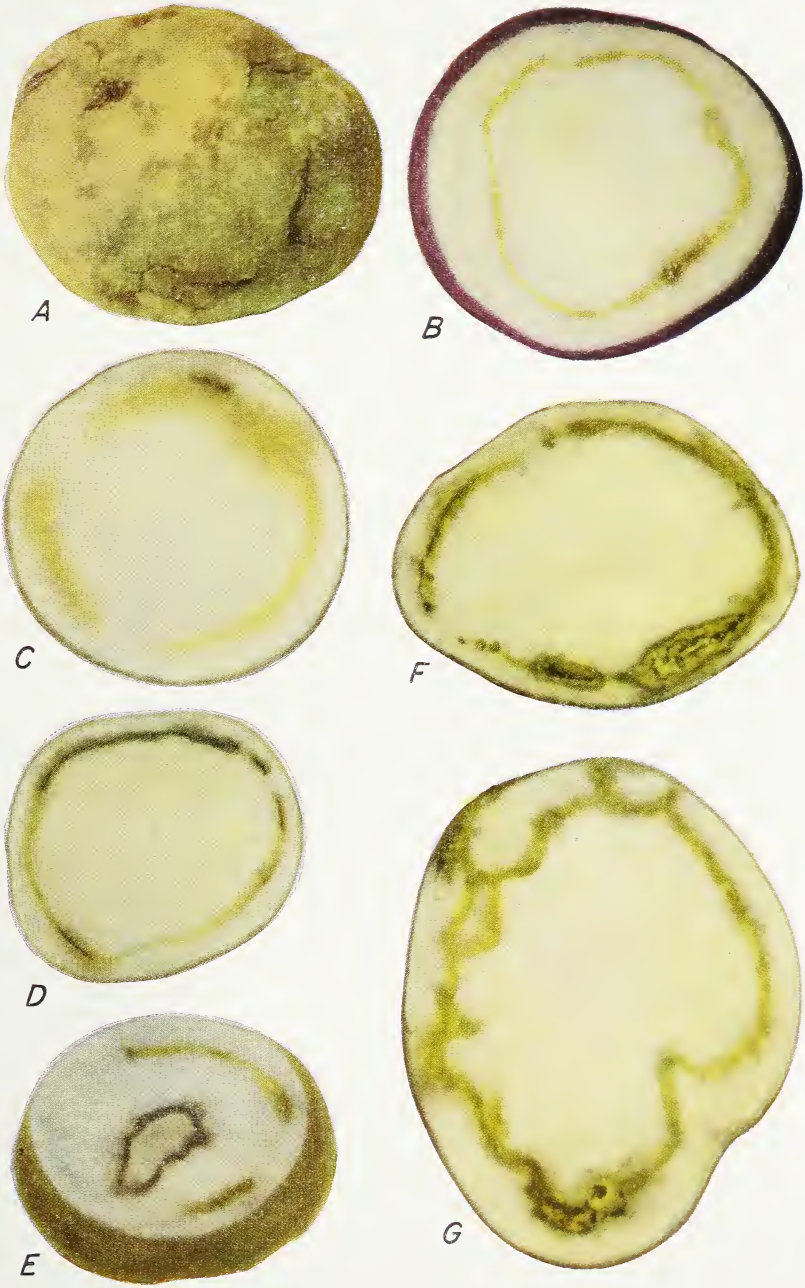
CONTROL MEASURES

Although seed-potato treatments often give good control of rhizoctonia canker under some conditions, delays in emergence of sprouts and reduction in stand and in yields due to the treatment make doubtful the desirability of treating seed stock used for growing table stock. However, potatoes used in seed plots should be treated. In some regions rotation of crops may prove of some value in reducing the amount of *Rhizoctonia* in the soil.

(See *19, 40, 73, 76, 112, 113, 153, 154, 162, 163, 164, 175, 176, 180, 198.*)



Potato diseases: *A*, Sand scurf; *B*, rhizoctonia black scurf; *C*, russeting caused by *Rhizoctonia*. *D*, Tuber showing characteristic russeting of the Russet Burbank type of potato.



Potato tubers affected with ring rot. *A*, External symptoms. *B–G*, Internal symptoms: *B*, Early stage; *C* and *D*, intermediate stages; *E–G*, advanced stages.

RING ROT

(*Corynebacterium sepedonicum* (Spieck. and Kotth.) Skapt. and Burkh.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Ring rot (bacterial ring rot), known in Germany since 1904–6, was found in Quebec, Canada, in 1931 and in Maine in 1932. Since then it has been reported from every important potato-producing State in this country.

Potentially it is one of the most serious diseases of potatoes, chiefly because of the ease and rapidity with which it can increase in amount within a given lot of potatoes during even a single growing season. It presents a very difficult problem for the grower of certified seed.

The seriousness of this disease is particularly difficult for inspectors at shipping points to estimate, because they are unable to detect it without first cutting the tubers. On the terminal markets tubers affected with ring rot are commonly found, and occasionally they are responsible for important market losses. Extensive decay due to secondary invasion of soft rot bacteria is also an important factor in market losses.

The symptoms of ring rot ordinarily do not appear in the field until late in the growing season; at that time the presence of other vine diseases or insect injuries may mask the ring rot symptoms. Affected plants first show a slight wilting of individual leaflets, leaves, stalks, or even the entire vine. This is accompanied by an upward rolling of the leaflets. Wilting is more pronounced during the warmer part of the day. Affected leaflets develop a marginal yellowing as their color changes from normal to pale green and finally to pale yellow. Brown, dead areas then appear, usually at the margins of the leaflets.

The chief symptom of ring rot on tubers is a characteristic odorless decay that is confined at first to the immediate vicinity of the vascular ring (pl. 23, *B*). This can readily be seen by slicing the tuber crosswise near the stem end. The infected vascular ring and adjacent flesh have a cream or pale lemon-yellow color that frequently approaches the color found in the normal ring of a healthy tuber (pl. 23, *B* and *C*). However, the decayed tissues are soft and of a cheesy consistency (pl. 23, *C* and *G*). They can readily be forced out of the ring by squeezing the tuber or by gently pressing a blunt pencil point against the vascular ring at the exposed surface of the tuber. In more severely affected tubers the diseased tissue can readily be pushed aside, leaving a narrow cavity or cleft that extends down into the tuber and runs part way or entirely around the ring (pl. 23, *D* and *F*).

Originating at the stolon attachment, the decay may progress uniformly in the vascular tissue or to nearby tissues. In this manner it may reach the surface of the tuber in the region of the eyes. This leads to a characteristic cracking that extends into the ring (pl. 23, *A*). Secondary soft rot bacteria (p. 5) may then enter and spread through the vascular ring and eventually affect the entire tuber (pl. 23, *E*). The presence of secondary rots may give rise to a variety of discolorations in the vicinity of the ring and may mask the symptoms of ring rot. A comparison of ring rot symptoms with those of brown rot (southern bacterial wilt) is found in the discussion of the latter disease (p. 9).

CAUSAL FACTORS

Corynebacterium sepedonicum, the bacterial pathogen which induces ring rot, lives over from season to season in the tubers. When infected tubers are planted, the bacteria move out into the developing sprouts and through the channels of the vascular ring they reach all parts of the growing plant. Later in the season the bacteria migrate from the stem out through the stolons, infecting the new tubers and thus perpetuating the disease from year to year.

The bacteria are readily transmitted from diseased tubers to healthy seed pieces during the cutting of the seed tubers. Likewise they may be spread during the planting operation, particularly if a picker-point type of planter is used. Another means by which the pathogen may be disseminated is through physical contact of the seed potatoes with infested bins, crates, baskets, tools, and other infested objects.

The bacteria do not overwinter in the soil, although an occasional infected tuber which survives from the previous crop may give rise to diseased volunteer plants. There is little if any evidence of spread from plant to plant in the field.

The period of incubation within the tuber may extend to 2 or 3 months. Consequently, infected tubers that show no visible evidence of the disease at harvest may develop positive symptoms in storage. Although healthy tubers can be infected through fresh wounds that penetrate as far as the vascular ring, there is ordinarily little if any spread from diseased to healthy tubers during the storage period.

The pathogen develops most rapidly at 68° to 74° F. A temperature range of 64° to 72° is said to be most favorable for the development of ring rot in stems, stolons, and tubers. Other tests have been reported in which infected tubers stored for 5 months developed ring rot most rapidly at 70° to 95°, less rapidly at 60° to 65°, and only slightly at 37°.

No commercially important varieties of potato are resistant to ring rot. Two foreign varieties and several seedling selections have shown a high degree of resistance. The disease has been transmitted experimentally to tomatoes, peppers, and eggplants.

CONTROL MEASURES

The most important means for preventing ring rot is to plant tubers that are free of the disease. Certified seed potatoes with zero tolerance for ring rot would be the best, since they could be used for a seed-potato plot or for the entire field.

After a crop in which ring rot has occurred has been harvested and marketed, but before the new seed lot is handled, the containers, tools, and implements of all kinds such as planters, diggers, and sorters used in handling the diseased crop should be thoroughly disinfected with solution of 1:1,000 mercuric chloride, 1:120 formaldehyde, or copper sulfate (2 pounds to 10 gallons). Likewise cellars and storage houses should be carefully disinfected in order to avoid contaminating new seed potatoes.

Unfortunately it is difficult to be sure that a given seed stock is entirely free of ring rot. For that reason it is advisable to take sanitary precautions, in case a small amount is present, to avoid any possible increase during the growing season. It should be kept in

mind constantly that ring rot is highly infectious and that it is spread by contact. Much of the spread occurs during the cutting of the seed. The knife should therefore be disinfected after cutting each tuber. Better still use a rotary knife which passes constantly through a suitable disinfectant such as boiling water. The use of an assist-feed planter is preferable to that of a picker-point type. The development of resistant varieties offers some promise for eventual control of the disease.

Special methods for maintaining disease-free seed lots include, among others, the examination of tubers during cutting at low temperatures under ultraviolet light and careful hill selection in seed plots planted with presumably disease-free seed potatoes combined with a rigid program of sanitation in building up seed-increase plots.

From a marketing standpoint it is advisable that lots known to contain any appreciable amount of ring rot be moved early and promptly. This may avoid increased losses from advanced stages of ring rot and from secondary bacterial soft rot that frequently follows (p. 5). The temperature recommended (38° F.) for potatoes in cold storage will control ring rot satisfactorily (p. 2).

(See *22, 27, 32, 33, 51, 57, 86, 98, 102, 104, 152, 182, 185, 190, 192, 193.*)

ROOT KNOT (NEMATODE GALL) [13]

(*Heterodera marioni* (Cornu) Goodey)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Root knot is a field disease of a very large number of wild and cultivated plants including most of our garden and truck-crop plants. The disease is common in greenhouses everywhere; in the field it occurs in all parts of the United States except the most northern States. It is particularly abundant in the sandy soils of the Gulf and Atlantic Coastal Plain regions as far north as Maryland. It has been reported on potatoes from almost all sections where they are grown commercially. Tubers affected with root knot are occasionally found on the market.

As indicated by the name, the chief symptom of root knot is the formation of thickened roots that are deformed by the presence of numerous knotlike galls. This results in dwarfed plants and reduced yields.

Potato tubers affected with advanced stages of root knot have a roughened, irregular, bumpy, or warty appearance due to the presence of galls on the outer surface (pl. 18, *C*). The skin over the galls usually remains unruptured. When such tubers are cut open the female nematodes are seen in abundance scattered through the outer tissues, with most of them approximately ⅛ to ¼ inch beneath the tuber surface. They appear as whitish bodies about the size of a small pinhead, each surrounded by a thin layer of brownish potato tissue (pl. 18, *B*). The infected parts of the tubers thus have a somewhat discolored appearance with some slightly water-soaked spots beneath the galls. Tubers sometimes show no external symptoms of root knot.

[13] Not to be confused with diseases caused by the golden nematode (p. 19) and the potato rot nematode (p. 34).

CAUSAL FACTORS

The organism that induces root knot is a nematode, or eelworm (*Heterodera marioni*). Between crop seasons it maintains itself in either the egg or the eelworm stage in the soil or in debris of infected plants. It also lives over in infected tubers. Preparasitic larvae and adult males can move through the soil as well as through infected plant tissues. When they come to the roots, stolons, or developing tubers, they bore their way into these plant organs, become sedentary, and begin to enlarge as they absorb nourishment. The female enlarges enormously in diameter so that she eventually becomes pear-shaped and large enough to be visible to the unaided eye; the male remains eel-shaped and not visible to the naked eye. The presence of the nematodes causes an irritation of the tissues as a result of which they develop abnormally and form the galls.

Larvae hatching from eggs within the tuber may move only a short distance within the tuber and continue to develop there; others move out into the soil and thus find their way to other roots or tubers. On Long Island, N. Y., it has been shown that there are usually 3 generations a year—1 on the roots and 2 on the tubers. In warm regions there may be as many as 10 to 12 generations in 1 year. Inasmuch as females are known to lay 500 to 2,000 eggs, it can readily be seen how rapidly the organism can multiply under favorable conditions. Soil temperature is an important factor. Most rapid development occurs at about 80° F.; below 50° to 55° the nematodes are relatively inactive. Certain races, however, can survive northern winters.

CONTROL MEASURES

The most important control measures are those designed to prevent further spread of the nematodes. Potatoes should never be used for seed purposes if they are known to have been produced on infested land. Care should be taken to avoid introducing the nematodes into uninfested soil through the application of garbage, manure, or waste material that might contain parts of diseased tubers or roots. Similar precautions should be used to avoid introducing the nematode through the planting of diseased seedlings or nursery stock of any kind. It should also be kept in mind that infested soil can be spread from field to field on machinery and tools, by irrigation and drainage water, and on the feet of man and domestic animals.

Once the nematodes have become established in a field it is difficult and costly to eradicate them. A 3-year rotation with nonsusceptible crops has been successful. Clean fallowing is an effective control measure. On Long Island a 1-year summer fallow materially reduced the infestation, and a 2-year fallowing practically eliminated the nematodes.

Where land is generally infested a soil-fumigation treatment with one of the newer fumigants such as dichloropropene or ethylene dibromide mixtures is recommended.[14]

(See *42*, *43*, *66*, *135*, *199*, *200*.)

[14] Marketed under such trade names as Shell D–D, Dowfume N, Dowfume W, Isocobrome D, Bromofume, and Soilfume.

RUSSETING

In addition to the natural russeting of some varieties of potatoes like the Russet Burbank (pl. 22, *D*) there are skin blemishes and discolorations of other varieties known as soil scurf, sand scurf, russet scab, and russeting. In many instances these blemishes are somewhat similar to the russeting induced by growth of *Rhizoctonia* on the surface of tubers (pl. 22, *C*) or to the superficial lesions caused by the common scab organism below. The exact cause of many of these skin blemishes is not known. The fact that by careful examination some *Rhizoctonia* can be found on practically all tubers has suggested that this fungus may be the cause of most of these troubles, but some studies have indicated that other causes may be responsible.

A very serious type of russeting, or scurf, that sometimes occurs in Missouri, Kansas, and Nebraska potatoes is illustrated in plate 22, *A*. No causal organism has been demonstrated, but soil treatments with some sterilizing agents have reduced the amount of this scurf, which indicates that an organism may be responsible for the trouble. Because this injury is usually most prevalent in sandy soils, it has been referred to as sand scurf; however, it is sometimes also found in heavier soils. It is usually most serious in soil that has previously been planted to potatoes.

Anything that irritates or injures the skin of growing tubers may lead to scurfy or scabby eruptions and corky discolorations that might be called russeting. Under some conditions tubers produced in contact with fertilizers in the soil may have rough skins that resemble russeting.

None of these types of skin blemishes lead to decay. However, the marketability of seriously russeted tubers is greatly influenced by their appearance.

(See *56*, *163*.)

SCAB

(*Actinomyces scabies* (Thaxt.) Güssow)

OCCURRENCE, SYMPTOMS, AND EFFECTS

The common scab of potatoes is universally distributed throughout the potato-growing regions of the United States. Although ordinarily yields are little affected the losses from reduced marketability of the crop may be very serious, for not only are scabby tubers unsightly but their paring wastage may exceed the tolerance for the U. S. No. 1 grade. Severely scabbed tubers are usually unsalable. Although some varieties of potatoes show a degree of resistance to scab, none is immune.

The first symptom of scab is the appearance of small reddish-brown spots on the surface of the young tuber. The formation of corky tissue over these spots as they enlarge and develop is the most characteristic symptom of scab (pl. 12, *C* and *D*). It is from the scablike appearance of this corky tissue that the disease gets its name. Considerable variation may occur, however, in the appearance of the scab lesions. Occasionally the surface may be only slightly roughened or russeted. (See also Rhizoctonia Black Scurf, p. 35, and Russeting, above.) Usually it is either raised in the form of rough, corky scabs

or sunken and in the form of shallow pits that are lined with the corky incrustations. The scab lesions may be in the form of isolated more or less rounded spots of various sizes, or they may coalesce and form large scabby areas on the tuber. Surface cracking occurs particularly when the lesions are extensive.

Scab symptoms are sometimes modified by the activities of insects and related forms. Thus where surface feeding by larvae of flea beetle (*Epitrix* spp.) occurs, infection by the scab pathogen may readily follow, so that the pattern of the scab lesions on the tuber is identical with that of the so-called "worm tracks." It is commonly believed that mites, scab gnat larvae, millepedes, and other animals feeding in scab pustules are in part responsible for the deep or pitted type of scab. Other investigators have reported that when these animals are present they feed only on dead tissue.

CAUSAL FACTORS

The causal agent of scab is *Actinomyces scabies,* one of the so-called higher, or filamentous, bacteria. Considerable variation occurs between isolates, indicating the presence of physiologic races within the species.

The pathogen, which is a common soil inhabitant, has been reported from virgin soils. Infection, which occurs chiefly through the lenticels of young tubers, may thus arise either from the organisms already present in the soil or from those introduced with scabby seed pieces at planting time. The pathogen does not attack mature tubers; consequently, after harvest there is no further progress of the disease on infected tubers and there is no spread of the disease to healthy tubers.

A number of different factors affect the development of the scab pathogen in the soil and thus determine the prevalence of the disease. Chief among these is the alkalinity or acidity of the soil. The development of scab is inhibited by acid soil reactions of about pH 5 to 5.2 or lower and by alkaline soil reactions of about pH 8 or higher. Greatest development has been found to occur between pH 5.45 and pH 7.4. Ordinarily low soil moisture favors scab development, but in some well-aerated soils (muck, for example) scab may be prevalent even with high soil moisture. It has been noted that scab is more severe during warm seasons and in those sections usually having high temperatures during the growing season. In controlled experiments scab developed throughout a rather wide range of soil temperatures but was most prevalent between 70° and 73° F. In pure culture the pathogen made little or no growth either below 41° or above 105°; best growth occurred between 77° and 86°.

CONTROL MEASURES

There is no certain method of controlling scab. Treatments that are helpful in one area may in other areas either be impractical or may give little promise of control. In general, the use of scabby seed should be avoided. Seed treatments should be employed if scab is present on the seed. These include dips with yellow oxide of mercury or commercial organic mercury preparations, and soaking in or dipping with formaldehyde solutions. In certain restricted areas good results

have been obtained by applying calomel or yellow oxide of mercury to the soil. The use of flowers of sulfur or inoculated sulfur as a soil amendment to increase the acidity has been a successful means of control in light soils. Results of potato-breeding investigations indicate considerable promise for the eventual control of scab through the development of scab-resistant varieties.

(See *1*, *20*, *38*, *39*, *45*, *71*, *91*, *96*, *100*, *111*, *116*, *117*, *125*, *131*, *158*, *173*, *174*, *177*.)

SCLEROTIUM ROT (SOUTHERN BLIGHT)

(*Sclerotium rolfsii* Sacc.[15])

OCCURRENCE, SYMPTOMS, AND EFFECTS

On the market sclerotium rot is frequently found affecting early-crop potatoes from the Southern States. In occasional years it is of major importance on potatoes from certain areas.

The rot first appears in the form of small, slightly sunken spots with brownish margins. These infections may take place anywhere on the tubers, but frequently they occur at the stem end (pl. 17, *A* and *B*) and at lenticels (pl. 17, *C*). Later the spots become more deeply sunken and yellowish or tan. At first the affected tissues, which are usually odorless and colorless, are firm; but later they become soft and mushy (pl. 17, *D*). In the more advanced stages the internal decayed tissues, which are then yellowish, shrink and collapse; at the same time the outer cortical tissues and skin become broken and fall away, thus forming sunken pockets in the flesh. Stem-end infections often result in the formation of moderately dry or spongy pockets of brown tissue extending into the pith region of the tuber (pl. 17, B).

The presence of the causal fungus itself is a ready aid in the identification of the disease. A rather fine, silky, white mycelium grows out over the surface of the lesions and adjacent healthy tissues (pl. 17, *A* and *E*). This is closely appressed to the surface and develops radially from a given point and forms fans. Grouping of the mold filaments into coarser strands is characteristic. Later, numerous small, rounded resting bodies, or sclerotia, develop on and in the mycelium. At first the sclerotia are whitish; but later they darken so that in color, size, and shape they closely resemble radish or mustard seeds (pl. 17, *A* and *E*). Both mycelium and sclerotia likewise develop in the pockets and cavities of the older lesions.

CAUSAL FACTORS

The pathogen, *Sclerotium rolfsii*, is a soil fungus that is widely distributed in the Southern States, where it attacks not only the potato but a great variety of truck crops and other plants as well. Infection usually occurs at or near the surface of the soil. Stem girdling with consequent vine wilting may take place; the main roots may be rotted, or fleshy plant parts near the surface of the soil may become infected and decayed.

[15] Perfect stage *Corticium rolfsii* (Sacc.) Curzi.

The fungus appears to thrive best in well-aerated sandy soil or loam. Development is favored by periods of wet weather. Optimum temperatures for growth of the pathogen are in the range of 86° to 95° F.; below 68° growth is poor. Undoubtedly these relatively high temperature requirements largely determine the geographical occurrence of the disease.

CONTROL MEASURES

Little can be done to control the disease in the field. Market losses can be reduced or avoided by maintaining transit temperatures below 68° F. and by moving the potatoes promptly.

(See *55*, *83*, *197*, *203*.)

SILVER SCURF

(*Spondylocladium atrovirens* Harz)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Silver scurf is a tuber disease that is found wherever potatoes are grown. Although occasionally of considerable local importance it ordinarily causes little more than a skin blemish and is generally overlooked on the market.

The occurrence of superficial brownish spots more or less circular in outline constitutes the first symptom of the disease on the tuber. The spots are not conspicuous on dry tubers, but when the tubers are wet they have a characteristic silvery appearance—hence the name. Lesions vary in shape and size, and at times they may cover most of the tuber surface (pl. 12, *A* and *B*). The lesions become darker with age. Living cells beneath the skin are not attacked; however, with the continued growth of the pathogen in the dead corky layers of the skin, the outer cells loosen and slough off. This permits increased water loss during storage so that when large areas of the tuber are affected the skin becomes shriveled, wrinkled, and in some cases actually folded.

CAUSAL FACTORS

Silver scurf is caused by *Spondylocladium atrovirens*, a fungus that lives from season to season on the tuber and in the soil. Apparently most of the disease originates from spores and mycelium borne in silver scurf lesions on infected seed pieces. It likewise appears that most of the first, or primary, cycles of infection occur in the field after the tubers have matured and while they lie in the soil before harvest. Spread of the disease from infected to healthy tubers may later take place in the field, but usually it occurs in the bin. This is particularly true in storage cellars where humidity is high and temperatures remain relatively high for some time after digging. Under humid conditions the causal fungus develops over the lesions and forms a fine blackish mold, which consists chiefly of the spores and spore-bearing stalks of the pathogen. The pathogen makes most rapid growth at temperatures of about 70° to 80° F. The formation of new lesions and the further enlargement of old ones are prevented by temperatures below 37° and by relative humidities below 90 percent.

CONTROL MEASURES

Seed-potato treatments have not been effective against silver scurf. Apparently the most efficient method for reducing the prevalence of the disease is to dig the tubers promptly after they have matured.

(See *31, 132, 178.*)

STEM-END BROWNING

Stem-end browning is a tuber disorder that has been of chief importance in Maine where it affects Green Mountain, Irish Cobbler, and a few less common varieties. As indicated by the name, the disease is recognized as a dark-brown or blackish discoloration that develops at the stem end of the tuber. When a thin slice is removed from the stem end of affected tubers, the discoloration appears in the center of the exposed surface where it is confined to the vascular elements that radiate from the point of the stolon attachment (pl. 16, *C*). Ordinarily the discoloration does not extend deeper than ¼ to ½ inch; at times, however, it does penetrate somewhat farther into the tuber. Such advanced cases present symptoms that are practically indistinguishable from those of the early stages of net necrosis (p. 31). Stem-end browning does not affect the vigor of the plant or the yield.

The cause of stem-end browning is unknown. Its occurrence cannot be correlated with soil type or with such soil factors as acidity, amount of organic matter, or availability of nutrients. There is some evidence that its occurrence is correlated with the frequency with which potatoes are grown on the same piece of land. Likewise, there are suggestions that chlorides in the fertilizer are a definite stimulus to the development of the trouble. There is rather definite evidence that the causal agent may be a virus.

Experiments in Maine have shown that there is a marked relation between storage temperature and the development of stem-end browning in susceptible tubers. Maximum development occurs when tubers are stored for about 100 days at 50° F. Less browning develops at either higher or lower temperatures. The first few weeks of storage is the most important period. It has been found that by conditioning susceptible potatoes for 60 days at either 70° or 32°, subsequent development of stem-end browning is practically inhibited. A temperature of 36° was nearly as effective as 32°. When varieties such as Katahdin and Chippewa, which are not susceptible to stem-end browning, are stored with Green Mountain, it is advisable to maintain the higher temperature (36°) because these newer varieties are susceptible to mahogany browning (p. 30) when stored at 32°.

Recent studies indicate that there are marked heritable differences in susceptibility between different commercial stocks or strains of the Green Mountain variety. In general, the susceptible strain had two to seven times as much stem-end browning as the more resistant strains. The Keswick strain was found highly susceptible. The use of one of the more resistant strains will partly control the trouble in strains of the Green Mountain variety. Unfortunately all Irish Cobbler lots tested have been found so susceptible that control of the disease in this variety can be accomplished only by the manipulation of storage temperatures in the manner described previously.

(See *60, 62, 63, 126, 127, 128, 129, 130, 169, 170.*)

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TRANSIT BRUISING

Potato tubers affected with transit bruising are commonly found on the market. Although occasional slight injury may occur when potatoes are pressed and rubbed together in the sack, most transit bruising occurs when the sacks come in contact with the floor racks and the walls of the car.

The injury is caused by the pressure of the load, by the shifting of bags at points of contact with the car, and by the jolting and jarring that occur while the car is in motion.

On newly harvested, early-crop potatoes the injured tissues are flattened or slightly sunken and are usually conspicuously darkened. When the bruised tissues are black (pl. 9, *C* and *D*), they frequently are mistaken for chemical injury by members of the trade. However, transit bruising can readily be distinguished from chemical injury (pl. 9, *A* and *B*) by the facts that there is no external softness or flabbiness of tissues beyond the borders of the flattened areas and that the tissues deeper than the flattened bruise do not turn black when the tuber is cut.

Injured tissues of late-crop tubers (pl. 24, *A* and *B*) are likewise flattened or slightly sunken, but they are seldom markedly discolored. When the bruises are cut through transversely, the injured areas are seen as a shallow layer of crushed tissue seldom more than ⅛ inch thick. At first the bruised layer is moist and somewhat tough; as it dries out it becomes firm and starchy. In some instances deeper tissues contain cracks that radiate from the bruise. Such shatter-bruise types of injury are readily apparent.

Occasionally in winter shipments transit bruising is mistaken for freezing injury. The two can readily be distinguished by a critical examination of affected tubers. Bruising symptoms are confined to the immediate vicinity of the flattened areas, but freezing may be recognized externally by softening or wrinkling of the flesh beyond the limits of the bruise and internally by softening or discoloration of tissues that occurs well beyond the border of the bruise (p. 15 and pl. 13). A tuber showing both transit bruising and transit freezing is shown in plate 24, *C* and *D;* in this tuber the frozen tissues can readily be distinguished from bruised ones. Symptoms of freezing in this tuber consist chiefly of softness of flesh without discoloration.

Transit bruising can be reduced by attention to the loading method, by more careful stowage of the load, and by using pads on the floor racks.

(See *165, 210.*)

VASCULAR BROWNING

"Vascular browning" is a descriptive term commonly used on the market to designate a slight brownish discoloration of the water-conducting tissues of the potato tuber. Normally these tissues, when exposed by slicing the tuber crosswise, appear as a faint circle inside the tuber a little way in from the skin, and forming the so-called vascular ring. When browning occurs, the ring becomes so conspicuous that the tuber may be considered somewhat damaged for food. Vascular browning is the least important of all tuber troubles that affect the vascular tissues. In appearance it most closely resembles very mild symptoms of fusarium wilt when the entire ring has been

A and *B*, Transit bruising on old potato tubers: *A*, External symptoms; *B*, internal symptoms on potato shown in *A*. *C* and *D*, Transit bruising followed by transit freezing on old potato tubers: *C*, Internal symptoms; *D*, external symptoms on potato shown in *C*.

only slightly discolored. Actually any one of several different troubles may be responsible for the browning. Observations have shown that vascular browning may develop during the storage period. It is usually found in greater abundance at the stem end of the tuber than elsewhere.

VERTICILLIUM WILT

(*Verticillium albo-atrum* Reinke and Berth.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

The symptoms of verticillium wilt are very similar to those produced by *Fusarium oxysporum* (pl. 16, *B;* p. 18). The disease stunts or kills the young plants, thus reducing the yield. So far as known the fungus does not cause decay of the tuber tissues, but it does cause discoloration of the vascular elements of the tubers. This lowers their market value and renders them unfit for sale as seed potatoes.

The first evidence of the disease in the growing plant is the loss of bright-green color and a tendency of the leaves to curl upward along the midrib, thus exposing their lower sides. The lower leaves are affected first and drop after wilting. Affected plants are frequently stunted, but they may not die for some time. Longitudinal or cross sections of stems or petioles of affected plants show the vascular ring to be brown or black, the discoloration often extending from the small rootlets, through the stem, and into the petioles. The discoloration often extends from the affected plant through the stolon into the tuber. The wilting of the plants is attributed to the plugging of the water-conducting vessels by the causal fungus. There does not appear to be any breaking down of tissues such as occurs occasionally in plants affected with fusarium wilts. At times a grayish mycelial growth, which distinguishes this disease from fusarium wilt, appears on the wilted stems at ground level.

CAUSAL FACTORS

Verticillium albo-atrum, the fungus that induces verticillium wilt, is one of the soil fungi. Infection of the growing plant can proceed either from infected potatoes produced by diseased plants the previous season or from soil. The disease also may spread in the field from plant to plant. Tubers become infected from plants through the stolons.

There is no development or spread of the disease in transit or in storage. The distribution of the disease indicates that moderate temperatures favor the organism; at least the disease does not develop in extremely high temperatures. The minimum temperature at which the disease may be expected to occur is 41° F., the optimum 77°, and the maximum 86°.

CONTROL MEASURES

Verticillium wilt may be controlled by planting tubers that are not diseased and using certified seed. The best way to avoid diseased tubers is to rogue the diseased plants in the seed plot. It has been stated that in Oregon a rotation of 5 years or more will help in eliminating the fungus from the soil. Absence of stem-end discolorations

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does not guarantee absence of the fungus; on the other hand, discoloration does not necessarily mean that this fungus is present any more than it does that *Fusarium* spp. is present.

(See *4, 54, 119, 150, 171.*)

VIRUS DISEASES

The potato is subject to attack by a wide variety of virus diseases. These include several different mosaics, leaf roll, spindle tuber, witches'-broom, yellow dwarf, and a number of others. Each is caused by a specific virus or by a combination of 2 viruses. Weiss [16] listed 14 typical potato viruses and 10 other viruses that, although infectious to potatoes, are typically identified with other plants. If the various strains of certain viruses are included the number responsible for diseases of the potato becomes much greater.

Virus particles are extremely small and are considered nonliving protein substances. However, when a susceptible plant is inoculated with only a small quantity of virus the latter multiplies so rapidly that later it may be found in great amount throughout the entire plant. In the more important virus diseases of potato the causal virus is spread from infected to healthy plants through the activities of aphids (plant lice) and other insects feeding on the foliage. They are likewise transmitted to the growing plant through planting of infected seed tubers.

Symptoms vary greatly with the several diseases. Those more frequently observed are mottling, wrinkling, curling, stunting, or distortion of leaves. Dwarfing and partial death of parts of the vine may also occur. Other symptoms are a stiff, upright appearance of the plant or an abnormal development of numerous slender shoots.

Although crop yields and the production of marketable tubers are greatly reduced by most of the virus diseases, only for a few of them are tuber symptoms apparent on the market. The most important of these symptoms is net necrosis (p. 31). Yellow dwarf (p. 49) is another.

Spindle tuber is likely to be found on the market in potatoes from certain sections. Tubers affected with this disease are characteristically spindle-shaped or cylindrical with the ends, particularly the bud end, abnormally long and pointed. The more numerous eyes are shallower, but they appear more conspicuous because of prominent eyebrows. There is frequently a fading of color on red-skinned varieties. Tubers affected with unmottled curly dwarf, a closely related disease, are irregular or spindle-shaped and often gnarled and cracked.

Potato plants infected with the tomato spotted wilt virus sometimes show necrotic spotting, cracking, and distortion of tubers. A brownish discoloration of the tuber flesh at the stem end is reported in some varieties affected with purple-top wilt.

In two diseases, tuber blotch and Canada streak, infected tubers may show the presence of internal discolored blotchy spots. With tuber blotch some of these necrotic areas may appear externally as dark

[16] Weiss, F. viruses, virus diseases, and similar maladies of potatoes, solanum tuberosum l. U. S. Bur. Plant Indus., Soils, and Agr. Engin., Plant Dis. Rptr. Sup. 155, pp. 82–140. 1945. [Processed.]

sunken lesions. Neither disease has been reported from the United States. Similarly, the veinal mosaic virus may cause both superficial and internal necrotic areas in the tubers of certain foreign varieties of potatoes.

(See *9, 48, 209.*)

WART

(*Synchytrium endobioticum* (Schilb.) Perc.)

Potato wart became known in the United States in 1918, when it was found in garden plots of a number of villages in the hard-coal districts of eastern Pennsylvania. It has since been found in small scattered districts of western Pennsylvania, western Maryland, and western Virginia. The disease originated with infected tubers imported from Europe. Because of strict quarantine regulations it is extremely unlikely that diseased tubers will be encountered on the market. The potential seriousness of wart makes it imperative, however, that all field and market inspectors familiarize themselves with the symptoms of the disease.

Wart first appears as small spongy outgrowths on the tuber particularly at the eyes (pl. 20, *A* and *B*). At first the warts are light brown in color. With age they enlarge, darken, and eventually become black. The warts may vary from the size of a pea to that of an entire tuber. Either few or many warty outgrowths may occur on a potato. If infection takes place early, the entire tuber may be converted into a worthless, spongy mass; other underground parts of the plant may likewise become infected.

The disease ordinarily does not affect the appearance of the stalks or foliage. It may originate with resting spores found in the diseased tissues of infected seed pieces or from soil that is infested with the resting spores of the pathogen. During periods of high soil moisture resting spores germinate and attack the growing potato plant. Infection can occur throughout a wide temperature range. Resting spores may remain viable in the soil for as long as 20 years. There is no development or spread of the disease in transit or in storage.

Rigid enforcement of quarantine measures has prevented the spread of wart from the general districts where it was first discovered. The use of resistant or immune varieties has been employed within these districts. During recent years it has been found that soil treatments with ammonium thiocyanate or with flaked copper sulfate will kill the fungus in infested soils. It appears that a continuation of the wart-eradication program with these materials may eventually completely eliminate the disease in this country.[17]

(See *44, 80, 85, 145, 204, 206.*)

YELLOW DWARF

Potatoes affected with yellow dwarf are seldom found on the market. Although not all tubers produced on diseased vines show external symptoms of the disease, most of them show malformations and cracks, which make it possible to grade out the affected tubers before shipment. Internal discolorations also make them unsuitable for table use.

[17] DYKSTRA, T. P. THE POTATO WART ERADICATION PROGRAM IN PENNSYLVANIA. U. S. Bur. Plant Indus., Plant Dis. Rptr. 24: 7–8. 1940. [Processed.]

The tubers produced on plants having yellow dwarf are usually small, irregular, knobby, and cracked (pl. 20, *D*). The cracks, which are ⅛ to ¼ inch deep, often radiate from the bud end and look like growth cracks. Such cracks usually heal without infection by decay-producing organisms. When diseased tubers are cut, rusty-brown specks and spots scattered in the pith area and extending from the bud end halfway to the stem are characteristic symptoms (pl. 20, *E*). Discolorations are seldom found at the stem end.

Yellow dwarf gets its name from the dwarfed condition and yellow color of affected plants in the field. The tips, or growing apexes, of diseased plants die early and the stalks soon become yellowish green, especially under high-temperature conditions. When diseased stalks are cut the characteristic rusty-brown specks like those within the tubers may be found in the pith and cortex. Seriously affected plants die before any tubers are formed.

Since the causal virus is carried over winter in insects such as the clover leafhopper, the planting of disease-free seed potatoes some distance from clover fields is suggested as one means of controlling the spread of this trouble.

(See *8, 14, 15, 16, 78, 201.*)

LITERATURE CITED

(1) Afanasiev, M. M.
1937. comparative physiology of actinomyces in relation to potato scab. Nebr. Agr. Expt. Sta. Res. Bul. 92, 63 pp.

(2) Artschwager, E. F.
1920. pathological anatomy of potato blackleg. Jour. Agr. Res. 20: 325–330, illus.

(3) ———
1927. wound periderm formation in the potato as affected by temperature and humidity. Jour. Agr. Res. 35: 995–1000, illus.

(4) Ayers, G. W., and Hurst, R. R.
1939. verticillium wilt of potatoes in prince edward island. Sci. Agr. 19: 722–735, illus.

(5) Bailey, F. D.
1914. notes on potato diseases from the northwest. Phytopathology 4: [321]–322, illus.

(6) Baker, A. D.
1946. the potato-rot nematode, ditylenchus destructor thorne, 1945, attacking potatoes in prince edward island. Sci. Agr. 26: 138–139.

(7) Barger, W. R., Ramsey, G. B., Perry, R. L., and MacGillivray, J. H.
1942. handling and shipping tests with new potatoes from kern county, california. Calif. Agr. Expt. Sta. Bul. 664, 24 pp., illus.

(8) Barrus, M. F., and Chupp, C. C.
1922. yellow dwarf of potatoes. Phytopathology 12: [123]–132, illus.

(9) ——— Chupp, C., and Fernow, K. H.
1940. potato diseases and their control. N. Y. Agr. Col. (Cornell) Ext. Bul. 135, 121 pp., illus. (Revised.)

(10) Bartholomew, E. T.
1913. black heart of potatoes. Phytopathology 3: [180]–182, illus.

(11) ———
1915. a pathological and physiological study of the black heart of potato tubers. Centbl. f. Bakt. [etc.] (Abt. II) 43: 609–639, illus.

(12) Bennett, J. P., and Bartholomew, E. T.
1924. the respiration of potato tubers in relation to the occurrence of blackheart. Calif. Agr. Expt. Sta. Tech. Paper 14, 40 pp., illus.

(13) Bergey, D. H., Breed, R. S., Murray, E. G. D., and others.
1939. bergey's manual of determinative bacteriology. . . . Ed. 5, 1032 pp. Baltimore, Md.
(14) Black, L. M.
1934. the potato yellow dwarf disease. Amer. Potato Jour. 11: 148–152.
(15) ———
1937. a study of potato yellow dwarf in new york. N. Y. (Cornell) Agr. Expt. Sta. Mem. 209, 23 pp., illus.
(16) ———
1941. specific transmission of varieties of potato yellow-dwarf virus by related insects. Amer. Potato Jour. 18: 231–233.
(17) Bliss, D. E.
1946. the relation of soil temperature to the development of armillaria root rot. Phytopathology 36: 302–318, illus.
(18) Blodgett, E. C., and Ray, W. W.
1945. leak, caused by pythium debaryanum hesse, produces typical "shell rot" of potato in idaho. Amer. Potato Jour. 22: 250–253, illus.
(19) Blodgett, F. M.
1939. the effects of some agronomic practices on the incidence of rhizoctonia. Amer. Potato Jour. 16: 93–98.
(20) ——— and Howe, F. B.
1934. factors influencing the occurrence of potato scab in new york. N. Y. (Cornell) Agr. Expt. Sta. Bul. 581, 12 pp., illus.
(21) Bonde, R.
1929. physiological strains of alternaria solani. Phytopathology 19: 533–548, illus.
(22) ———
1939. bacterial wilt and soft rot of the potato in maine. Maine Agr. Expt. Sta. Bul. 396, pp. [675]–694, illus.
(23) ———
1939. comparative studies of bacteria associated with potato blackleg and seed-piece decay. Phytopathology 29: 831–851.
(24) ———
1939. the role of insects in the dissemination of potato blackleg and seed-piece decay. Jour. Agr. Res. 59: 889–917, illus.
(25) ——— and Schultz, E. S.
1943. potato refuse piles as a factor in the dissemination of late blight. Maine Agr. Expt. Sta. Bul. 416, pp. [229]–246, illus.
(26) ——— Stevenson, F. J., and Clark, C. F.
1940. resistance of certain potato varieties and seedling progenies to late blight in the tubers. Phytopathology 30: 733–748, illus.
(27) ——— Stevenson, F. J., Clark, C. F., and Akeley, R. V.
1942. resistance of certain potato varieties and seedling progenies to ring rot. Phytopathology 32: 813–819.
(28) Brien, R. M.
1941. "leak," a watery wound-rot of potatoes in new zealand. New Zeal. Jour. Sci. and Technol., A, 22: 228A–231A, illus.
(29) Brierley, P.
1928. pathogenicity of bacillus mesentericus, b. aroideae, b. carotovorus, and b. phytophthorus to potato tubers. Phytopathology 18: 819–838, illus.
(30) Bruyn, H. L. G. de.
1926. the overwintering of phytophthora infestans (mont.) de by. Phytopathology 16: 121–140, illus.
(31) Burke, O. D.
1938. the silver-scurf disease of potatoes. N. Y. (Cornell) Agr. Expt. Sta. Bul. 692, 30 pp., illus.
(32) Burkholder, W. H.
1938. the occurrence in the united states of the tuber ring rot and wilt of the potato (phytomonas sepedonica) (spickermann u. kotthoff) bergey et al. Amer. Potato Jour. 15: 243–245.

(33) ———
1942. DIAGNOSIS OF THE BACTERIAL RING ROT OF THE POTATO. Amer. Potato Jour. 19: 208–212.
(34) CAIRNS, H., and MUSKETT, A. E.
1933. PINK ROT OF THE POTATO. Ann. Appl. Biol. 20: 381–403, illus.
(35) ——— and MUSKETT, A. E.
1939. PHYTOPHTHORA ERYTHROSEPTICA PETHYBR. IN RELATION TO ITS ENVIRONMENT. Ann. Appl. Biol. 26: 470–480, illus.
(36) CARPENTER, C. W.
1915. SOME POTATO TUBER-ROTS CAUSED BY SPECIES OF FUSARIUM. Jour. Agr. Res. 5: 183–210, illus.
(37) CHITWOOD, B. G., and BUHRER, E. M.
1946. THE LIFE HISTORY OF THE GOLDEN NEMATODE OF POTATOES, HETERODERA ROSTOCHIENSIS WOLLENWEBER, UNDER LONG ISLAND, NEW YORK, CONDITIONS. Phytopathology 36: 180–189, illus.
(38) CLARK, C. F., RALEIGH, W. P., and STEVENSON, F. J.
1936. BREEDING FOR RESISTANCE TO COMMON SCAB IN THE POTATO. Amer. Potato Jour. 13: 256–259.
(39) ——— STEVENSON, F. J., and SCHAAL, L. A.
1938. THE INHERITANCE OF SCAB RESISTANCE IN CERTAIN CROSSES AND SELFED LINES OF POTATOES. Phytopathology 28: 878–890, illus.
(40) CLARK, E. S., and MARTIN, W. H.
1931. THE EFFECT OF DEPTH OF PLANTING AND SOIL MOISTURE ON THE DEVELOPMENT OF RHIZOCTONIA ON THE POTATO. (Abstract) Phytopathology 21: 105.
(41) CROSIER, W.
1934. STUDIES IN THE BIOLOGY OF PHYTOPHTHORA INFESTANS (MONT.) DE BARY. N. Y. (Cornell) Agr. Expt. Sta. Mem. 155, 40 pp., illus.
(42) CUNNINGHAM, H. S.
1936. THE ROOT-KNOT NEMATODE (HETERODERA MARIONI) IN RELATION TO THE POTATO INDUSTRY ON LONG ISLAND. N. Y. State Agr. Expt. Sta. Bul. 667, 24 pp., illus.
(43) ——— and MAI, W. F.
1947. NEMATODES PARASITIC ON THE IRISH POTATO. N. Y. (Cornell) Agr. Col. Ext. Bul. 712, 24 pp., illus.
(44) CURTIS, K. M.
1921. THE LIFE-HISTORY AND CYTOLOGY OF SYNCHYTRIUM ENDOBIOTICUM (SCHILB.), PERC., THE CAUSE OF WART DISEASE IN POTATO. Roy. Soc. London, Phil. Trans., Ser. B, 210: 409–478, illus.
(45) DARLING, H. M.
1937. A STUDY OF SCAB RESISTANCE IN THE POTATO. Jour. Agr. Res. 54: 305–317.
(46) DAVIS, W. B.
1926. PHYSIOLOGICAL INVESTIGATION OF BLACK HEART OF POTATO TUBER. Bot. Gaz. 81: 323–338, illus.
(47) DRECHSLER, C.
1929. A DIPLANETIC SPECIES OF PHYTOPHTHORA CAUSING PINK ROT OF POTATO TUBERS. (Abstract) Phytopathology 19: 92.
(48) DYKSTRA, T. P.
1948. POTATO DISEASES AND THEIR CONTROL. U. S. Dept. Agr. Farmers' Bul. 1881, 53 pp., illus. (Revised.)
(49) EDDINS, A. H.
1936. BROWN ROT OF IRISH POTATOES AND ITS CONTROL. Fla. Agr. Expt. Sta. Bul. 299, 44 pp., illus.
(50) ———
1939. ADJUSTING pH REACTIONS OF SOILS WITH SULFUR AND LIMESTONE TO CONTROL BROWN ROT OF POTATOES. Amer. Potato Jour. 16: [6]–16, illus.
(51) EDDINS, A. H.
1939. SOME CHARACTERISTICS OF BACTERIAL RING ROT OF POTATOES. Amer. Potato Jour. 16: [309]–322, illus.
(52) ——— PROCTOR, E. Q., and WEST, E.
1946. CORKY RINGSPOT OF POTATOES IN FLORIDA. Amer. Potato Jour. 23: 330–333, illus.

(53) Edmundson, W. C., and Schaal, L. A.
1940. potato breeding for fusarium resistance. Amer. Potato Jour. 17: 92–95, illus.
(54) Edson, H. A., and Shapovalov, M.
1920. temperature relations of certain potato-rot and wilt-producing fungi. Jour. Agr. Res. 18: 511–524, illus.
(55) ——— and Shapovalov, M.
1923. parasitism of sclerotium rolfsii on irish potatoes. Jour. Agr. Res. 23: 41–46, illus.
(56) [Elmer, A. H.]
1944. potato production in kansas: part ii.—diseases and their control. Kansas Agr. Expt. Sta. Bul. 322, pp. 26–44, illus.
(57) Fernow, K. H.
1944. potato ring rot control for those who think they don't have the disease. Amer. Potato Jour. 21: 14–17.
(58) Folsom, D.
1921. potato leafroll. Maine Agr. Expt. Sta. Bul. 297, pp. [37]–52, illus.
(59) ———
1933. botrytis cinerea as a cause of potato tuber rot. Phytopathology 23: 993–999, illus.
(60) ———
1946. leafroll net necrosis and stem-end browning of potato tubers in relation to temperature and certain other factors. Phytopathology 36: 1016–1034.
(61) ——— and Bonde, R.
1925. alternaria solani as a cause of tuber rot in potatoes. Phytopathology 15: [282]–286, illus.
(62) ——— Libby, W. C., Simpson, G. W., and Wyman, O. L.
1938. net necrosis of the potato. Maine Agr. Col. Ext. Bul. 246, 12 pp., illus.
(63) ——— and Rich, A. E.
1940. potato tuber net-necrosis and stem-end browning studies in maine. Phytopathology 30: 313–322, illus.
(64) Gilbert, A. H.
1927. net necrosis of the potato. Phytopathology 17: 555–561, illus.
(65) ———
1928. net-necrosis of irish potato tubers. Vt. Agr. Expt. Sta. Bul. 289, 36 pp., illus.
(66) Godfrey, G. H.
1923. root-knot: its cause and control. U. S. Dept. Agr. Farmers' Bul. 1345, 27 pp., illus.
(67) Goss, R. W.
1921. temperature and humidity studies of some fusaria rots of the irish potato. Jour. Agr. Res. 22: 65–80, illus.
(68) ———
1924. potato wilt and stem-end rot caused by fusarium eumartii. Nebr. Agr. Expt. Sta. Res. Bul. 27, 83 pp., illus.
(69) ———
1936. fusarium wilts of potato, their differentiation and the effect of environment upon their occurrence. Amer. Potato Jour. 13: 171–180.
(70) ———
1937. the fusarium wilt of potatoes. Ohio Veg. Growers' Assoc. Proc. 22: 60–66.
(71) ———
1937. the influence of various soil factors upon potato scab caused by actinomyces scabies. Nebr. Agr. Expt. Sta. Res. Bul. 93, 40 pp., illus.
(72) Goss, R W.
1940. a dry rot of potato stems caused by fusarium solani. Phytopathology 30: 160–165, illus.
(73) ——— and Afanasiev, M. M.
1938. influence of rotations under irrigation on potato scab, rhizoctonia, and fusarium wilt. Nebr. Agr. Expt. Sta. Bul. 317, 18 pp., illus.

(74) ——— and Jensen, J. H.
1941. varietal susceptibility of potatoes to fusarium wilt. Amer. Potato Jour. 18: 209–212.
(75) ——— and Jensen, J. H.
1944. a pythiaceous stem-end rot of potatoes. (Abstract) Phytopathology 34: 1001.
(76) Gratz, L. O.
1930. effect of seed-potato treatment on yield and rhizoctonosis in florida from 1924 to 1929. Fla. Agr. Expt. Sta. Bul. 220, 34 pp., illus.
(77) ——— and Bonde, R.
1927. infection of potato tubers by alternaria solani in relation to storage conditions. Fla. Agr. Expt. Sta. Bul. 187, pp. [165]–182, illus.
(78) Hansing, E. D.
1943. a study of the control of the yellow-dwarf disease of potatoes. N. Y. (Cornell) Agr. Expt. Sta. Bul. 792, 28 pp., illus.
(79) Harding, H. A., and Morse, W. J.
1910. the bacterial soft rots of certain vegetables. part i. the mutual relationships of the causal organisms. Vt. Agr. Expt. Sta. Bul. 147: [241]–279. (Also N. Y. [State] Agr. Expt. Sta. Tech. Bul. 11, pp. [250]–287. 1909.)
(80) Hartman, R. E., and Akeley, R. V.
1944. potato wart in america. Amer. Potato Jour. 21: 283–288.
(81) Hatfield, W. C., and Kreutzer, W. A.
1943. careful handling of potatoes is of prime importance in avoiding loss from dry rot. Colo. Farm Bul. 5 (5): 3–4, illus.
(82) Hawkins, L. A.
1916. the disease of potatoes known as "leak." Jour. Agr. Res. 6: 627–640, illus.
(83) Higgins, B. B.
1927. physiology and parasitism of sclerotium rolfsii sacc. Phytopathology 17: 417–448, illus.
(84) Hilborn, M. T., and Bonde, R.
1942. a new form of low-temperature injury in potatoes. Amer. Potato Jour. 19: 24–29, illus.
(85) Hunt, N. R., O'Donnell, F. G., and Marshall, R. P.
1925. steam and chemical soil disinfection with special reference to potato wart. Jour. Agr. Res. 31: 301–363, illus.
(86) Iverson, V. E., and Kelly, H. C.
1940. control of bacterial ring rot of potatoes with special reference to the ultraviolet-light method for selecting disease-free seed stock. Mont. Agr. Expt. Sta. Bul. 386, 15 pp., illus.
(87) Jensen, J. H., and Livingston, J. E.
1945. potato diseases in nebraska. Nebr. Agr. Expt. Sta. Bul. 378, 47 pp., illus.
(88) Jones, A. P., and Moore, H. I.
1935–36. cracking of potato tubers. Gard. Chron. 98: 445–446, illus., 1935; 99: 185, illus., 1936.
(89) [Jones, L. R.]
1901. a soft rot of carrot and other vegetables caused by bacillus carotovorus, jones. Vt. Agr. Expt. Sta. Ann. Rpt. (1899–1900) 13: 299–332, illus.
(90) Jones, L. R., Giddings, N. J., and Lutman, B. F.
1912. investigations of the potato fungus phytophthora infestans. U. S. Bur. Plant Indus. Bul. 245, 100 pp., illus.
(91) Jones, L. R., McKinney, H. H., and Fellows, H.
1922. the influence of soil temperature on potato scab. Wis. Agr. Expt. Sta. Res. Bul. 53, 35 pp., illus.
(92) ——— Miller, M., and Bailey, E.
1919. frost necrosis of potato tubers. Wis. Agr. Expt. Sta. Res. Bul. 46, 46 pp., illus.

(93) Jones, W.
1935. soft rot of potatoes caused by pythium ultimum trow. Sci. Agr. 15: 402–410, illus.
(94) ———
1945. pink rot disease of potatoes in british columbia. Sci. Agr. 25: 597–600, illus.
(95) ——— and MacLeod, H. S.
1937. armillaria dry rot of potato tubers in british columbia. Amer. Potato Jour. 14: 215–217, illus.
(96) KenKnight, G.
1941. studies on soil actinomycetes in relation to potato scab and its control. Mich. Agr. Expt. Sta. Tech. Bul. 178: 48 pp.
(97) Klaus, H.
1940. untersuchungen über alternaria solani jones et grout, insbesondere über seine pathogenität an kartoffelknollen in abhängigkeit von den aussenfaktoren. Phytopath. Ztschr. 13: [126]–195, illus.
(98) Knorr, L. C.
1944. efficacy of the rotary knife in the control of potato ring rot. Amer. Potato Jour. 21: 250–261.
(99) Kotila, J. E., and Coons, G. H.
1925. investigations on the blackleg disease of potato. Mich. Agr. Expt. Sta. Tech. Bul. 67, 29 pp., illus.
(100) Krantz, F. A., and Eide, C. J.
1941. inheritance of reaction to common scab in the potato. Jour. Agr. Res. 63: 219–231.
(101) Kreutzer, W. A., and Lane, G. H.
1945. new potato disease of leak type found in colorado; measures for control listed. Colo. Farm Bul. 7 (2): 3–4, illus.
(102) ——— and McLean, J. G.
1943. location and movement of the casual agent of ring rot in the potato plant. Colo. Expt. Sta. Tech. Bul. 30, 28 pp., illus.
(103) Kunkel, L. O.
1915. a contribution to the life history of spongospora subterranea. a preliminary report. Jour. Agr. Res. 4: 265–278, illus.
(104) Larson, R. H.
1944. the ring rot bacterium in relation to tomato and eggplant. Jour. Agr. Res. 69: 309–325, illus.
(105) ——— and Albert, A. R.
1945. physiological internal necrosis of potato tubers in wisconsin. Jour. Agr. Res. 71: 487–505, illus.
(106) Leach, J. G.
1926. the relation of the seed-corn maggot (phorbia fuscieps zett.) to the spread and development of potato blackleg in minnesota. Phytopathology 16: [149]–176, illus.
(107) ———
1927. the nature of seed-piece transmission of potato blackleg. Phytopathology 17: 155–160, illus.
(108) ———
1930. potato blackleg: the survival of the pathogene in the soil and some factors influencing infection. Phytopathology 20: 215–228, illus.
(109) ———
1930. the identity of the potato blackleg pathogene. Phytopathology 20: 743–751.
(110) ———
1931. blackleg disease of potatoes in minnesota. Minn. Agr. Expt. Sta. Tech. Bul. 76, 36 pp., illus.
(111) Leach, J. G., Decker, P., and Becker, H.
1939. pathogenic races of actinomyces scabies in relation to scab resistance. Phytopathology 29: 204–209, illus.
(112) ——— Johnson, H. W., and Parson, H. E.
1929. the use of acidulated mercuric chloride in disinfecting potato tubers for the control of rhizoctonia. Phytopathology 19: 713–724, illus.

(113) LeClerg, E. L., Person, L. H., and Meadows, S. B.
1942. FURTHER STUDIES ON THE TEMPERATURE RELATIONS OF SCLEROTIAL ISOLATES OF RHIZOCTONIA SOLANI FROM POTATOES. Phytopathology 32: 731–732.
(114) Link, G. K. K.
1916. A PHYSIOLOGICAL STUDY OF TWO STRAINS OF FUSARIUM IN THEIR CAUSAL RELATION TO TUBER ROT AND WILT OF POTATO. Bot. Gaz. 62: 169–209, illus. (Reprinted as Nebr. Agr. Expt. Sta. Res. Bul. 9, 45 pp., illus. 1916.)
(115) ——— and Gardner, M. W.
1919. MARKET PATHOLOGY AND MARKET DISEASES OF VEGETABLES. Phytopathology 9: [497]–520.
(116) Lutman, B. F.
1913. THE PATHOLOGICAL ANATOMY OF POTATO SCAB. Phytopathology 3: [255]–264, illus.
(117) ———
1941. ACTINOMYCES IN POTATO TUBERS. Phytopathology 31: 702–717, illus.
(118) McCubbin, W. A.
1946. GOLDEN NEMATODE AS A QUARANTINE PROBLEM. (Abstract) Phytopathology 36: 687.
(119) McKay, M. B.
1921. TRANSMISSION OF SOME WILT DISEASES IN SEED POTATOES. Jour. Agr. Res. 21: 821–848, illus.
(120) McLean, J. G., and Walker, J. C.
1941. A COMPARISON OF FUSARIUM AVENACEUM, F. OXYSPORUM, AND F. SOLANI VAR. EUMARTII IN RELATION TO POTATO WILT IN WISCONSIN. Jour. Agr. Res. 63: 495–525, illus.
(121) MacLeod, G. F., and Rawlins, W. A.
1933. INSECT AND OTHER INJURIES TO POTATO TUBERS. N. Y. (Cornell) Agr. Expt. Sta. Bul. 569, 14 pp., illus.
(122) MacMillan, H. G.
1920. A FROST INJURY OF POTATOES. Phytopathology 10: [423]–424, illus.
(123) ———
1931. TURGESCENCE AND RUPTURE OF POTATO TUBER. (Abstract) Phytopathology 21: 701–703.
(124) ——— and Meckstroth, G. A.
1925. THE CRITICAL TEMPERATURE FOR INFECTION OF THE POTATO SEED PIECE BY FUSARIUM OXYSPORUM. Jour. Agr. Res. 31: 917–921.
(125) ——— and Schaal, L. A.
1929. A PATHOLOGICAL FEATURE OF FLEA-BEETLE INJURY OF POTATO TUBERS. Jour. Agr. Res. 39: 807–815, illus.
(126) Maine Agricultural Experiment Station.
1941. POTATOES. Maine Agr. Expt. Sta. Ann. Rpt. (Bul. 405) 57: 470–509, illus.
(127) ———
1942. POTATOES. Maine Agr. Expt. Sta. Ann. Rpt. (Bul. 411) 58: [281]–343, illus.
(128) ———
1943. POTATOES. Maine Agr. Expt. Sta. Ann. Rpt. (Bul. 420) 59: [419]–482, illus.
(129) ———
1944. POTATOES. Maine Agr. Expt. Sta. Ann. Rpt. (Bul. 426) 60: [211]–297, illus.
(130) ———
1945. POTATOES. Maine Agr. Expt. Sta. Ann. Rpt. (Bul. 438) 61: [505]–602, illus.
(131) Martin, W. H., and Daines, R. H.
1941. POTATO SCAB. N. J. Agr. Expt. Sta. Cir. 415, 10 pp.
(132) Melhus, I. E.
1913. SILVER SCURF, A DISEASE OF THE POTATO. U. S. Bur. Plant. Indus. Cir. 127, pp. 15–24, illus.
(133) ———
1915. GERMINATION AND INFECTION WITH THE FUNGUS OF THE LATE BLIGHT OF POTATO (PHYTOPHTHORA INFESTANS). Wis. Agr. Expt. Sta. Res. Bul. 37, 64 pp., illus.

(134) METCALF, C. L., and FLINT, W. P.
1939. DESTRUCTIVE AND USEFUL INSECTS, THEIR HABITS AND CONTROL. Ed. 2, 981 pp., illus. New York.
(135) MIDDLETON, J. T.
1940. RECENT ROOT-KNOT DAMAGE IN POTATOES. (Abstract) Phytopathology 30: 709.
(136) MILLS, W. R.
1940. PHYTOPHTHORA INFESTANS ON TOMATO. Phytopathology 30: 830–839.
(137) MOORE, H. C.
1927. HOLLOW HEART OF POTATOES. Mich. Agr. Expt. Sta. Quart. Bul. 9: 137–139.
(138) MORSE, W. J.
1914. POWDERY SCAB OF POTATOES. Maine Agr. Expt. Sta. Bul. 227, pp. [89]–104, illus.
(139) ———
1917. STUDIES UPON THE BLACKLEG DISEASE OF THE POTATO, WITH SPECIAL REFERENCE TO THE RELATIONSHIP OF THE CAUSAL ORGANISMS. Jour. Agr. Res. 8: 79–126.
(140) MURPHY, P. A.
1936. SOME EFFECTS OF DROUGHT ON POTATO TUBERS. Empire Jour. Expt. Agr. 4: 230–246, illus.
(141) ——— and MCKAY, R.
1924. THE DEVELOPMENT OF BLIGHT IN POTATOES SUBSEQUENT TO DIGGING. Ireland Dept. Lands and Agr. Jour. 24: 103–116.
(142) ——— and M'KAY, R.
1927. SOME FURTHER CASES OF THE PRODUCTION OF DISEASED SHOOTS BY POTATO TUBERS ATTACKED BY PHYTOPHTHORA INFESTANS, AND A DEMONSTRATION OF ALTERNATIVE SOURCES OF FOLIAGE AND TUBER INFECTION. Roy. Dublin Soc. Sci. Proc. (n. s.) 18: 413–422, illus.
(143) NIELSEN, L. W.
1946. SOLAR HEAT IN RELATION TO BACTERIAL SOFT ROT OF EARLY IRISH POTATOES. Amer. Potato Jour. 23: 41–57.
(144) ——— and TODD, F. A.
1946. BACTERIAL SOFT ROT OF IRISH POTATOES AS INFLUENCED BY SUBLETHAL TEMPERATURES. Amer. Potato Jour. 23: [73]–87, illus.
(145) ORTON, C. R., and KERN, F. D.
1919. THE POTATO WART DISEASE. Pa. Agr. Expt. Sta. Bul. 156, 16 pp., illus.
(146) PEACOCK, W. M., WRIGHT, R. C., and WHITEMAN, T. M.
1932. THE RELATION OF SOLAR AND SKY RADIATION, TEMPERATURE, AND HUMIDITY TO THE SUN-SCALD OF POTATOES IN 1931. Amer. Soc. Hort. Sci. Proc. 28: 261–265.
(147) PETERS, E. J.
1943. STEM-END VASCULAR DISCOLORATION OF POTATOES DUE TO FUSARIUM OXYSPORUM, F. TUBEROSI. Amer. Potato Jour. 20: 10–12.
(148) PETHYBRIDGE, G. H.
1913–19. INVESTIGATIONS ON POTATO DISEASES. [PINK ROT AND WILT (PHYTOPHTHORA ERYTHROSEPTICA PETHYB).]. Ireland Dept. Agr. and Tech. Instr. Jour. 13: 461–465, illus., 1913; 14: 452–455, 1914; 15: 510–511, illus., 1915; 16: 596, 1916; 19: 281–282, 1919.
(149) ———
1916. INVESTIGATIONS ON POTATO DISEASES. (SEVENTH REPORT). Ireland Dept. Agr. and Tech. Instr. Jour. 16: 564–596.
(150) PETHYBRIDGE, G. H.
1916. THE VERTICILLIUM DISEASE OF THE POTATO. Roy. Dublin Soc. Sci. Proc. (n. s.) 15: 63–92, illus.
(151) PRATT, O. A.
1916. CONTROL OF THE POWDERY DRYROT OF WESTERN POTATOES CAUSED BY FUSARIUM TRICHOTHECIOIDES. Jour. Agr. Res. 6: 817–832, illus.

(152) Racicot, H. N., Savile, D. B. O., and Conners, I. L.
1938. bacterial wilt and rot of potatoes—some suggestions for its detection, verification, and control. Amer. Potato Jour. 15: 312–318, illus.
(153) Raleigh, W. P., and Bonde, R.
1936. seed-potato treatment for rhizoctonia control in northeastern Maine, 1929 to 1933. Phytopathology 26: 321–343.
(154) Ramsey, G. B.
1917. a form of potato disease produced by rhizoctonia. Jour. Agr. Res. 9: 421–426, illus.
(155) ———
1918. influence of moisture and temperature upon infection by spongospora subterranea. Phytopathology 8: 29–31.
(156) ———
1941. botrytis and sclerotinia as potato tuber pathogens. Phytopathology 31: 439–448, illus.
(157) ——— Lutz, J. M., Werner, H. O., and Edgar, A. D.
1944. experiments on shipping washed early potatoes. Nebr. Agr. Expt. Sta. Bul. 364, 32 pp., illus.
(158) Reddick, D.
1939. scab immunity. Amer. Potato Jour. 16: [71]–76.
(159) ———
1943. development of blight-immune varieties. Amer. Potato Jour. 20: 118–126.
(160) ——— and Mills, W.
1938. building up virulence in phytophthora infestans. Amer. Potato Jour. 15: [29]–34.
(161) ——— and Peterson, L. C.
1945. empire — a blight resistant variety. Amer. Potato Jour. 22: 357–362.
(162) Richards, B. L.
1921. pathogenicity of corticium vagum on the potato as affected by soil temperature. Jour. Agr. Res. 21: 459–482, illus.
(163) Rolfs, F. M.
1902. potato failures. a preliminary report. Colo. Agr. Expt. Sta. Bul. 70, 20 pp., illus.
(164) ———
1904. potato failures. a second report. Colo. Agr. Expt. Sta. Bul. 91, 33 pp., illus.
(165) Rose, D. H.
1946. handling and shipping early potatoes. U. S. Dept. Agr. Cir. 744, 44 pp., illus.
(166) ——— and Fisher, D. F.
1940. desiccation effects on skinned potatoes. Amer. Potato Jour. 17: [287]–289.
(167) ——— and Schomer, H. A.
1944. relation of heat and desiccation to bacterial soft rot of potatoes. Amer. Potato Jour. 21: [149]–161, illus.
(168) Rosenbaum, J., and Ramsey, G. B.
1918. influence of temperature and precipitation on the blackleg of potato. Jour. Agr. Res. 13: 507–513, illus.
(169) Ross, A. F.
1946. studies on the cause of stem-end browning in green mountain potatoes. Phytopathology 36: 925–936, illus.
(170) ———
1946. susceptibility of green mountain and irish cobbler commercial strains to stem-end browning. Amer. Potato Jour. 23: [219]–234, illus.
(171) Rudolph, B. A.
1931. verticillium hadromycosis. Hilgardia 5: [197]–360, illus.
(172) Ruehle, G. D.
1940. bacterial soft rot of potatoes in southern florida. Fla. Agr. Expt. Sta. Bul. 348, 36 pp., illus.
(173) Sanford, G. B.
1923. the relation of soil moisture to the development of common scab of potato. Phytopathology 13: [231]–236, illus.

(174) ———
1926. SOME FACTORS AFFECTING THE PATHOGENICITY OF ACTINOMYCES SCABIES. Phytopathology 16: 525–547, illus.
(175) ———
1938. STUDIES ON RHIZOCTONIA SOLANI KÜHN. III. RACIAL DIFFERENCES IN PATHOGENICITY. Canad. Jour. Res., Sect. C, 16: [53]–64, illus.
(176) ———
1938. STUDIES ON RHIZOCTONIA SOLANI KÜHN. IV. EFFECT OF SOIL TEMPERATURE AND MOISTURE ON VIRULENCE. Canad. Jour. Res., Sect. C, 16: 203–213, illus.
(177) SCHAAL, L. A.
1934. RELATION OF THE POTATO FLEA BEETLE TO COMMON SCAB INFECTION OF POTATOES. Jour. Agr. Res. 49: 251–258, illus.
(178) SCHULTZ, E. S.
1916. SILVER-SCURF OF THE IRISH POTATO CAUSED BY SPONDYLOCLADIUM ATROVIRENS. Jour. Agr. Res. 6: 339–350, illus.
(179) ——— and FOLSOM, D.
1921. LEAFROLL, NET-NECROSIS, AND SPINDLING-SPROUT OF THE IRISH POTATO. Jour. Agr. Res. 21: 47–80, illus.
(180) SHAPOVALOV, M.
1922. RHIZOCTONIA SOLANI AS A POTATO-TUBER ROT FUNGUS Phytopathology 12: 334–336, illus.
(181) ——— and EDSON, H. A.
1921. BLACKLEG POTATO TUBER-ROT UNDER IRRIGATION. Jour. Agr. Res. 22: 81–92, illus.
(182) SHERF, A. F.
1944. INFECTION EXPERIMENTS WITH POTATO RING ROT AND THE EFFECT OF SOIL TEMPERATURE ON THE DISEASE. Amer. Potato Jour. 21: [27]–29.
(183) SINGH, B. N., and MATHUR, P. B.
1937. NEGATIVE CORRELATION BETWEEN THE OCCURRENCE OF POLYPHENOL OXIDASE AND DIASTASE AND THE DEGREE OF INCIDENCE OF "BLACKHEART" OF POTATO. Phytopathology 27: 992–1000.
(184) ——— and MATHUR, P. B.
1938. ARTIFICIAL PRODUCTION OF "BLACKHEART" IN POTATO TUBERS. Phytopathology 28: 705–708.
(185) SKAPTASON, J. B.
1943. STUDIES ON THE BACTERIAL RING-ROT DISEASE OF POTATOES. N. Y. (Cornell) Agr. Expt. Sta. Mem. 250, 30 pp.
(186) SMITH, E. F.
1896. A BACTERIAL DISEASE OF THE TOMATO, EGGPLANT AND IRISH POTATO. (BACILLUS SOLANACEARUM N. SP.). U. S. Dept. Agr. Div. Veg. Phys. and Path. Bul. 12, 28 pp., illus.
(187) ———
1914. BROWN ROT OF SOLANACEAE. *In* Bacteria in Relation to Plant Diseases 3: 174–219, illus. (Carnegie Inst. Wash., Pub. 27.)
(188) ———
1920. AN INTRODUCTION TO BACTERIAL DISEASES OF PLANTS. 688 pp., illus. Philadelphia and London.
(189) SMITH, M. A., and RAMSEY, G. B.
1947. BACTERIAL LENTICEL INFECTION OF EARLY POTATOES. Phytopathology 37: 225–242, illus.
(190) SNIESZKO, S. F., and BONDE, R.
1943. STUDIES ON THE MORPHOLOGY, PHYSIOLOGY, SEROLOGY, LONGEVITY, AND PATHOGENICITY OF CORYNEBACTERIUM SEPEDONICUM. Phytopathology 33: 1032–1044, illus.
(191) STANFORD, E. E., and WOLF, F. A.
1917. STUDIES ON BACTERIUM SOLANACEARUM. Phytopathology 7: [155]–165, illus.
(192) STAPP, C.
1930. BEITRÄGE ZUR KENNTNIS DES BACTERIUM SEPENDONICUM SPIECKERM. ET KOTTH., DES ERREGERS DER "BAKTERIENRINGFÄULE" DER KARTOFFEL. Ztschr. f. Parasitenk. 2: [756]–823, illus.
(193) STARR, G. H., and RIEDL, W. A.
1941. BACTERIAL RING-ROT OF POTATOES. Wyo. Agr. Expt. Sta. Bul. 244, 12 pp., illus.

(194) STEINBAUER, G. P.
1945. TREATMENT OF POTATO REFUSE PILES TO PREVENT SPREAD OF LATE BLIGHT. Maine Agr. Expt. Sta. Bul. 434, pp. [286]–292, illus.
(195) STEVENSON, F. J., and CLARK, C. F.
1938. THE SEBAGO POTATO, A NEW VARIETY RESISTANT TO LATE BLIGHT. U. S. Dept. Agr. Cir. 503, 7 pp., illus.
(196) STEWART, F. C., and MIX, A. J.
1917. BLACKHEART AND THE AERATION OF POTATOES IN STORAGE. N. Y. [State] Agr. Expt. Sta. Bul. 436, pp. 321–362, illus.
(197) TAUBENHAUS, J. J.
1919. RECENT STUDIES ON SCLEROTIUM ROLFSII SACC. Jour Agr. Res. 18: 127–138, illus.
(198) THATCHER, F. S.
1942. A STEM-END ROT OF POTATO TUBERS CAUSED BY RHIZOCTONIA SOLANI. Phytopathology 32: 727–730, illus.
(199) THORNE, G.
1942. DISTRIBUTION OF THE ROOT-KNOT NEMATODE IN HIGH RIDGE PLANTINGS OF POTATOES AND TOMATOES. (Abstract) Phytopathology 32: 650.
(200) TYLER, J.
1933. THE ROOT-KNOT NEMATODE. Calif. Agr. Expt. Sta. Cir. 330, 35 pp., illus.
(201) WALKER, J. C., and LARSON, R. H.
1939. YELLOW DWARF OF POTATO IN WISCONSIN. Jour. Agr. Res. 59: 259–280, illus.
(202) WATSON, R. D.
1944. CHARCOAL ROT OF IRISH POTATOES. Phytopathology 34: 433–435, illus.
(203) WEBER, G. F.
1943. SOUTHERN BLIGHT, CORTICIUM ROLFSII, OF POTATO TUBERS. (Phytopath. note) Phytopathology 33: 615–617, illus.
(204) WEISS, F.
1925. THE CONDITIONS OF INFECTION IN POTATO WART. Amer. Jour. Bot. 12: 413–443, illus.
(205) ——— LAURITZEN J. I., and BRIERLEY, P.
1928. FACTORS IN THE INCEPTION AND DEVELOPMENT OF FUSARIUM ROT IN STORED POTATOES. U. S. Dept. Agr. Tech. Bul. 62, 36 pp., illus.
(206) ——— ORTON, C. R., and HARTMAN, R. E.
1923. INVESTIGATIONS OF POTATO WART. U. S. Dept. Agr. Dept. Bul. 1156, 22 pp., illus.
(207) WERNER, H. O.
1928. HOLLOW HEART OF POTATOES: OCCURRENCE AND TEST OF THIOUREA SEED TREATMENTS FOR PREVENTION. Potato Assoc. Amer. Proc. (1927) 14: 71–88, illus.
(208) WIANT, J. S.
1945. INTERNAL BLACK SPOT OF LONG ISLAND POTATO TUBERS. Amer. Potato Jour. 22: 6–11, illus.
(209) WILTSHIRE, S. P., ed.
1946. COMMON NAMES OF VIRUS DISEASES USED IN THE REVIEW OF APPLIED MYCOLOGY. Rev. Appl. Mycol. 24: [513]–556.
(210) WRIGHT, R. C.
1939. BRUISING, FREEZING, AND CHEMICAL INJURY OF POTATOES IN TRANSIT. U. S. Dept. Agr. Tech. Bul. 668, 23 pp., illus.
(211) ——— and DIEHL, H. C.
1927. FREEZING INJURY TO POTATOES. U. S. Dept. Agr. Tech. Bul. 27, 24 pp., illus.
(212) ——— and TAYLOR, G. F.
1921. FREEZING INJURY TO POTATOES WHEN UNDERCOOLED. U. S. Dept. Agr. Bul. 916, 15 pp., illus.

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